The New England JOURNAL of MEDICINE

FORMERLY THE BOSTON MEDICAL AND SURGICAL JOURNAL

Official Organ of The Massachusetts Medical Society
The New Hampshire Medical Society—The Vermont State Medical Society
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OWNED AND PUBLISHED BY THE MASSACHUSETTS MEDICAL SOCIETY

Volume 211 Number 11 | Domestic $6 per Annum, 25c per Copy Canada $7.04, Foreign $8.52 | SEPTEMBER 13, 1934 | Published Weekly in Boston at 8 Fenway

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The Massachusetts Medical Society

The next Annual Meeting of the Massachusetts Medical Society will be held in Boston, at the Hotel Statler, June 3, 4, and 5, 1935.

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The New England
Journal of Medicine

VOLUME 211 SEPTEMBER 13, 1934 NUMBER 11

AUTOPSY FINDINGS IN 100 CASES OF PROSTATIC CANCER*

BY E. ROSS MINTZ, M.D.,† AND GEORGE GILBERT SMITH, M.D.†

THE necropsies forming the basis for this paper were obtained from the pathologic departments of the Massachusetts General, Boston City and Palmer Memorial Hospitals, fifty-three cases from the Massachusetts General Hospital, twenty-seven from the Boston City Hospital and thirty from the Palmer Memorial Hospital. No attempt has been made to discuss the findings in their entirety or to draw any conclusions from them. The facts are presented as found, but in their evaluation one should bear in mind that, in most instances, carcinoma in an advanced stage was present. In only thirteen cases (see table 1) could the neoplastic lesion be considered early. Five of them were autopsy findings and the condition was unsuspected during life. In one instance microscopic section showed the supposed benign lesion to be malignant. This is not uncommon (Swan, R. H. J.[1], Wade[2], Bugbee[3], Hirsch and Schmidt[4], Wilson and McGrath[5], Randall[6], Eastes and Fletcher[7], Ferguson[8]). In the remaining seven cases the neoplastic process was confined to the prostate without any gross or microscopic evidence of metastasis.

TABLE 1

CASES OF EARLY CARCINOMA OF THE PROSTATE FOUND AT NECROPSY

	Visceral Metastasis	Glandular Extension	Osseous Metastasis	Renal and Ureteral Pathology	Extension to Vesicles	Age	
1	x	x	x	x	x	67	Entered for symptoms of portal obstruction and peripheral edema. Cancer found at autopsy.
2	x	x	x	x	x	69	Died of sepsis following suprapubic prostatectomy. Microscopic section showed cancer.
3	x	x	x	x	x	81	Died cardiac death; mural thrombi of left auricle. Cancer found at autopsy.
4	x	x	x	x	x	62	Lobular pneumonia; multiple brain abscesses. Cancer found at necropsy.
5	x	x	x	x	x	63	Pulmonary embolus; thrombophlebitis of iliac vein and vena cava. Early carcinoma.
6	x	x	x	x	x	73	Bronchopneumonia; acute cholecystitis and cholelithiasis; early carcinoma.
7	x	x	x	x	x	84	Lobular pneumonia; early carcinoma.
8	x	x	x	x	x	76	Pulmonary infarct. Cancer found at autopsy.
9	x	x	x	x	x	59	Lobar pneumonia; cardiac hypertrophy and dilatation; early carcinoma.
10	x	x	x	x	x	70	Cardiac death; ascites, anasarca; pulmonary infarction; early cancer.
11	x	x	x	x	x	74	Subdural hemorrhage; early carcinoma.
12	x	x	x	x	x	70	Infarction of ileum. Early cancer. Benign prostatic hypertrophy.
13	x	x	x	x	x	73	Radiation necrosis of bladder and prostate. Thrombosis of right iliac vein. Early cancer.

"x" signifies no pathology.

The histopathology of this series will be discussed in a later paper. There was one case in which an epidermoid carcinoma of the prostate was found. (See figure 1.) This is most uncommon (Volante[9], Neller and Neubürger[10], Kaufmann[11]). The microscopic sections that were reviewed from all the autopsies at the Massachusetts General Hospital and some from

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the Palmer Memorial Hospital showed perineurial invasion quite frequently. (See figure 2.) Ferguson[8] gives the incidence of nerve invasion as twenty-six per cent.

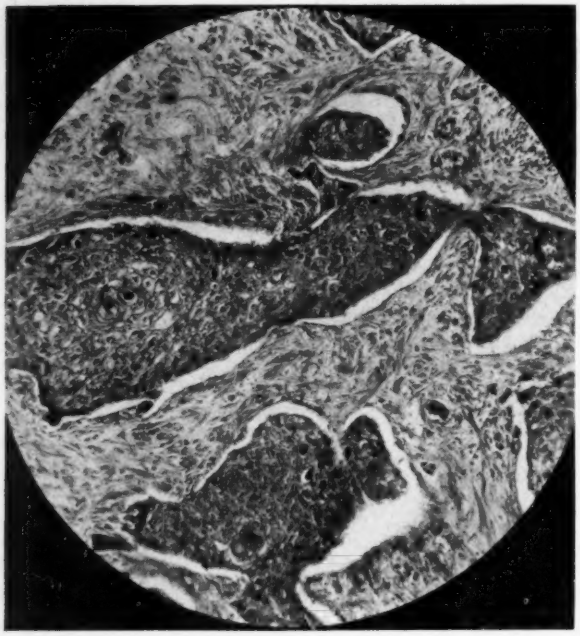

FIGURE 1. Epidermoid carcinoma of the prostate. Note tendency to cornification. (High power.)

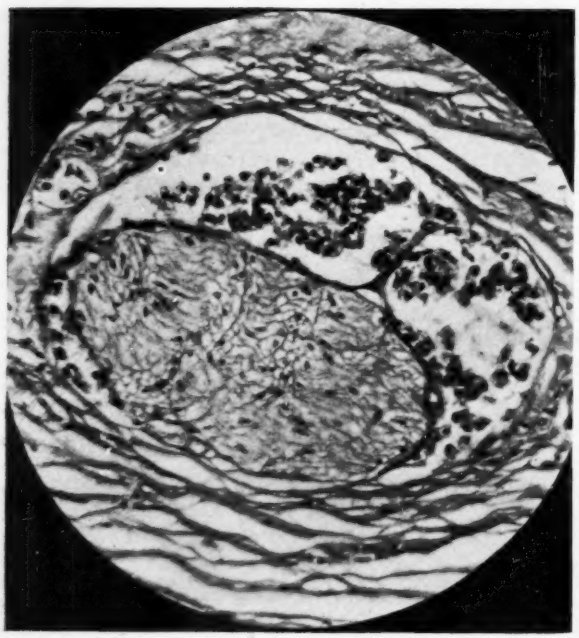

FIGURE 2. Invasion of nerve sheath in prostatic capsule. (High power.)

SEMINAL VESICLES

The seminal vesicles were the seat of cancerous spread fifty-eight times. In forty cases the bladder was jointly invaded and in fifteen cases the glands were invaded without joint invasion of the bladder. The vesicles were free from carcinomatous extension thirty-nine times and no mention was made of the pathology present in three instances. There was one case in which unilateral vesicular extension was noted. This is not surprising as most of the cases in this group were advanced cancer. The extraordinarily high incidence of vesicular extension in cancer of the prostate is nothing new. Young and Frontz[12] found this organ invaded in forty-two out of sixty-three patients operated upon perineally for malignant diseases of the prostate. In nineteen of them the neoplastic process was confined to only one half of the prostate and corresponding vesicle. Marion[13] cites vesicular invasion in forty-four (95 per cent) out of forty-six cases of total perineal prostatectomy and vesiculectomy for carcinoma of the prostate. Deming[14] observed the vesicles to be involved ninety times in 100 clinical cases. Dossot's[15] series show neoplastic extension to the vesicles in fifteen out of thirty-two necropsies. Motz and Majewski[16] reported invasion of the vesicles in twenty out of twenty-six postmortems and felt that the neoplastic masses are ordinarily more developed on the left side. This observation cannot be substantiated by this series as no mention was made in the autopsy protocols as to the relative invasion of each lobe. Wolff's[17] figures show the vesicles invaded thirteen times in sixty-four autopsies. The left vesicle alone was invaded seven times. There was one case in this group in which the primary lesion was thought to be in the seminal vesicles and not in the prostate. Although the prostate was the seat of cancer, yet the neoplastic process seemed to involve the vesicles to a greater extent. The metastases were soft, massive, extensive and in gross appearance were not the usual ones seen in prostatic cancer. It was definitely not a sarcoma or an interacinous (medullary) form of prostatic cancer. The microscopic picture was of little help, for both are glandular organs, and all that could be said was that it was an undifferentiated carcinoma. Whether it was vesicular or prostatic in origin still remains unsolved. So far as we are aware, an indisputable, proved case of primary carcinoma of the seminal vesicles has never been reported. There is no reason why it should not occur. Extension to the bladder was noted in fifty per cent of the cases. Thirty-nine times pelvic lymphadenopathy was associated with it and in forty cases the seminal vesicles and bladder were involved together. There were ten instances in which the bladder showed extension of the neoplastic process without seminal vesicle involvement and eleven cases of bladder extension with no pelvic lymphadenopathy.

BLADDER

Neoplastic infiltration of the musculature of the bladder base, trigone and bladder neck was the type of extension usually found. (See table 2.) Ulceration of the vesical mucosa was described in some cases. Fungating tumor masses, situated in the trigonal region and sometimes involving the bladder outlet, were noted in about eight per cent of the cases. The percentage of this type of bladder extension is low compared with the statistics of other authors (Motz and Majewski[16], Hallopeau[18], Thompson-Walker[19]). The intravesical tumor masses in this series were usually multiple, always sessile, rather firm and small, and almost always situated in the trigonal region. The mucosa covering these tumor nodules was ulcerated in some cases. Description of the pathology around the bladder neck was not carefully noted in most cases. However, there were some cases in which the vesical neck and prostatic urethra were so grossly invaded by neoplasm that the bladder outlet was almost completely occluded.

TABLE 2

EXTENSION TO BLADDER, LYMPH NODES, AND VESICLES

	Per Cent
Extension to bladder	50
Extension to bladder and lymph nodes	39
Extension to bladder without lymph node involvement	11
Extension to bladder and vesicles	40
Extension to bladder without vesicular involvement	10
Extension to vesicles	58
Extension to vesicles without bladder involvement	18
Extension to vesicles without bladder involvement but with extension to lymph nodes	15
Extension to pelvic or retroperitoneal nodes	.60
Extension to nodes without bladder or vesicular involvement	6

RECTUM

The incidence of propagation of the neoplastic process to the rectal wall and mucosa was noted in nine cases in this series. (See table 5.) In only one case was there any mention of rectal ulceration. This, of course, is valueless for in most instances a careful examination of the rectal mucosa was not made. In the majority of cases microscopic sections of the rectal wall were lacking and no notation was made as to the presence or absence of rectal pathology. The figures quoted in the literature for rectal extension from carcinoma of the prostate varied markedly. Young[20] reports "not more than a dozen cases" in a careful study of about 800 cases of prostatic cancer (1.5 per cent). Barringer[21] cites three instances of rectal invasion in 145 cases, in which the process extended through the rectal wall and produced a vesicorectal or ureterorectal fistula. Not one case of spontaneous vesico- or ureterorectal fistula was present in this series. Wolff's[17] statistics (cited by Jacobson) showed rectal extension in thirteen per cent (8 out of 64 cases), and Dossot's[15] 7.9 per cent (5 out of 63). Deming[14] found the rectal wall invaded in fifteen per cent of 100 cases. In not one case was mucosal ulceration present. It is felt that with a careful microscopic examination in all autopsied cases, the incidence of extension of the neoplastic process to the rectal musculature will approach more the figure quoted by Deming[14] and be even greater.

Rectal obstruction, due to the extrinsic prostatic mass, did not occur in this series. It has occurred, however. (Jacobson[22], Englisch[23].)

LYMPHADENOPATHY

In sixty cases, lymphatic extension to the retroperitoneal glands was present. (See table 3.) The pelvic, iliac and retroperitoneal nodes together were involved twenty-two times. There was only one case in which the iliac glands were invaded without concomitant invasion of the retroperitoneal chain. The preaortic glands showed cancer extension eight times, the bronchial and hilum glands nine times, mesenteric nodes seven times, axillary once, inguinals twice, mediastinal nodes without microscopic or gross lung involvement twice, and mediastinal nodes with lung extension three times. It is interesting to note the low incidence of superficial glandular involvement (2 per cent). This is probably due to failure of notation on the necropsy protocols; for in recently reviewing 200 cases of prostatic cancer, it was found that the inguinal glands were involved in the metastatic process thirteen times (6.5 per cent), the cervicals eleven times (5.5 per cent), axillaries four times (2 per cent), and the supraclaviculars twice (1 per cent).

TABLE 3

LYMPHADENOPATHY

	Number of Cases
Retroperitoneal glands	60
Retroperitoneal, iliac, pelvic	22
Pre-aortic	8
Bronchial	9
Mediastinal	5
Supraclavicular	1
Axillary	1
Mesenteric	7
Cervical	1
Inguinal	2

The incidence of lymphatic extension in prostatic cancer varies considerably with the report of different authors, but all statistics appear to be uniformly high. In seventy-one postmortems

Pasteau[24] reports iliac lymphadenopathy in eighty-seven per cent of the cases, retroperitoneal nodes in twenty-seven per cent and inguinal glands in thirty-six per cent. Bumpus[25] demonstrated metastases in 243 of 1000 clinical cases; of these forty-four per cent showed lymphatic involvement, inguinals in eighteen per cent (44 cases) and cervical glands in ten per cent (25 cases). Wolff[17] found the pelvic nodes invaded thirty-one times and the superficial glands six times in sixty-eight autopsied cases. Kaufmann[11] reported the pelvic lymphatics invaded twenty-seven times in 100 autopsies. Green, quoted by Bryan[26], found the retroperitoneal and inguinal glands invaded in eighty-five per cent of seventy-one autopsies on cancer of the prostate and the axillary nodes in five per cent. Young[27], in forty cases of perineal prostatectomy, cites invasion of the lymphatics eleven times. In seventeen necropsies, Randall[6] found extension to the pelvic nodes five times. Dossot[15] added thirty-eight autopsies showing pelvic extension in fifty-five and two-tenths per cent (21 cases), iliacs forty-four and seven-tenths per cent (17 cases), common iliacs fifty-five per cent (20 cases), retroperitoneal chain eighty-six and eight-tenths per cent (33 cases), inguinal nodes three times, and supraclavicular and tracheobronchial glands once each. Motz and Majewski's[16] incidence of lymphatic involvement in malignancy of the prostate is notoriously high, twenty-five out of twenty-six postmortems.

In only one instance was massive glandular involvement described (such as reported by Haszard[28] [5-6 cm.]). By and large, the glands were firmer than normal and discrete. Occasionally they were adherent in a solid retroperitoneal chain, but necrosis was uncommon. There were only a few cases in which the entire iliac, retroperitoneal chain, mediastinal and cervical glands were involved, but these were the exception rather than the rule. Masses of pelvic glands as described by Guyon[29] (carcinose prostato-pelvienne) were noted only a few times. Vascular compression due to these glandular masses, with edema of one or both extremities, was noted six times. Although the internal, external and common iliacs were frequently surrounded by neoplasm, invasion of their walls never occurred. The case of Chalier[30], in which neoplastic invasion and ulceration of the external iliac artery caused massive hemorrhage and death, is interesting in this respect. However, this complication is exceedingly rare. Thrombosis of the iliac vein, or of one of its tributaries, was present in seven per cent of the cases, but in only a few cases was neoplasm found within the vessel. (See figure 3.) The majority of them could be attributed to postoperative pelvic sepsis, or radiation, or both.

In one case the pelvic, iliac and bronchial lymph nodes were involved, and the retroperitoneal chain of nodes skipped. This "skipping" of nodes occurs not infrequently in carcinomata of other organs and just why this occurs is hard to state. Probably careful microscopic sections of all the glands along the retroperitoneal chain are not done. Compression of the bile ducts from without by a glandular mass and neoplastic invasion of the wall of the gall-bladder

FIGURE 3. High power view of invasion of one of the veins in the pelvis.

occurred in one case. In one of the two cases with inguinal node enlargement, the scrotum was edematous. This, clinically, is not an uncommon finding when the inguinal glands are involved, and ofttimes the edema recedes after a course of radiation over the inguinal region. There is very little doubt that, in cases in which inguinal lymphadenopathy exists, the neoplastic process is far advanced. Bumpus[25] reported a case in which an inguinal lymph node was removed, ten years after the initial diagnosis of carcinoma of the prostate had been made, and was found to be carcinomatous.

Extension to lymph nodes without bladder or vesicular invasion was present in only six cases, and these organs might have been found to be invaded if more microscopic sections had been done. It can then be stated, with a certain degree of safety, that if glandular invasion exists, either the vesicles or bladder are involved in the malignant process. One should continually bear in mind, however, that one is dealing here with necropsies and the disease in its late stages.

VISCERAL METASTASES

Visceral metastases from prostatic cancer were present twenty-four times in the lung and twen-

ty times in the liver. (See table 4.) In a few cases no mention was made of metastasis to these organs, as the autopsies were restricted. It should be noted that most of these cases were ones in which the neoplastic process was advanced or in which there was carcinosis. An incidence of twenty-four per cent of pulmonary metastasis, however, is unusually high, for clinically the percentage is much lower. In 200 clinical cases of prostatic cancer at the Massachusetts General Hospital roentgenograms of the chest were taken in 106. Of these only seven were positive for metastatic malignancy, or about seven per cent. There were a few instances in which the interpretation of the x-ray was negative for metastasis and small discrete nodules of cancer tissue were found at necropsy near the lung periphery beneath the pleura. In about half the cases showing pulmonary metastasis the bronchial or mediastinal glands were involved. When these glands were not involved, lymphadenopathy existed elsewhere. In a few cases there was microscopic evidence of tumor cells in the blood vessels and in four instances the pleura was involved. The subpleural lymphatics in a number of them were filled with tumor emboli. (See figure 4.) Pleural and pulmonary extension was noted twice in thirty autopsied cases by Motz and Majewski[16]. Jacobson[22] reported invasion of the pleura six times and lung four times in sixty-seven necropsies. Randall[6] found pleural and lung metastases in one out of seventeen postmortems. There was only one case in this group in which pulmonary metastasis was present without any gross evidence of glandular or visceral extension, exception being made of extradural metastases in this particular instance. This speaks strongly for a blood borne route of dissemination.

TABLE 4

INCIDENCE OF VISCERAL METASTASIS

	Number of Cases
Lung	24
Liver	20
Spleen	0
Kidney	3
Ureter	1
Suprarenal	1
Pancreas	1
Gall-Bladder	1
Pericardium	1
Diaphragm	1
Eye	1
Penis	1
Skin	1
Spinal cord and brain	4

The tumor nodules were for the most part small, firm, multiple and discrete. In no case were the nodules so large as those described by McKechnie[31] (1 cm.) and, except for cough, they did not give rise to any symptoms.

The liver was the seat of metastatic malignancy twenty times. In two cases the nodules were massive and in one of these jaundice was present, but it could be debated in this case whether the icterus was directly due to the diffuse hepatic metastases or to extrinsic pressure on the ducts from enlarged metastatic glands. Massive hepatic metastatic nodules are uncommon, but cases have been reported (Bonnamour and Giraud[32], Mann[33]), and in one instance the clinical signs were those of cirrhosis. However, these are most uncommon and the type usually seen, even in advanced cases, is small, multiple, firm and discrete. Solitary nodules do occur oftimes, but are for the most part associated with extension elsewhere. Jacobson[22] found liver metastasis six times in sixty-seven cases, Motz and Majewski[16] three times in thirty necropsies and Randall[6] failed to find a single instance of liver metastasis in seventeen cases. These statistics are much lower than the ones in this series.

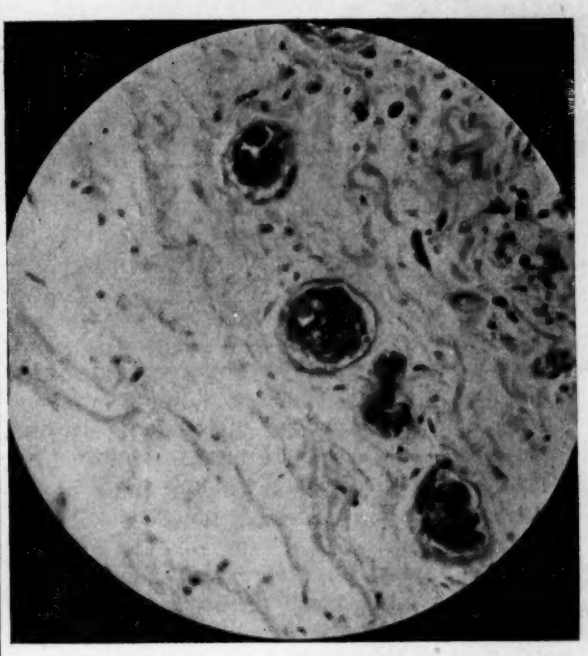

FIGURE 4. Low power view of tumor emboli in vessels beneath the pleura.

Peritoneal extension occurred three times. In all the cases there was gross evidence of mesenteric lymphadenopathy and in two of them massive glandular metastases were present, the largest measuring 1 cm. in diameter. As a general rule metastasis to the gastro-intestinal tract is usually associated with it, but in one instance the latter was not involved.

The gastro-intestinal tract, rectum excepted, was invaded in ten per cent of the cases; stomach three times, large intestine three times, esophagus twice, and jejunum and cecum once

each. (See table 5.) Mesenteric glands were enlarged and involved in most of them, and in no instance was intestinal obstruction present, nor were there any gastro-intestinal symptoms that could be definitely attributed to them. Metastasis to this tract usually occurs in very advanced cases of prostatic cancer. Invasion of the gall-bladder was noted once.

Splenic metastases did not occur in this series and are most uncommon. They have been noted, however, by others (Jacobson[22], Randall[6]).

TABLE 5

METASTASIS TO THE GASTRO-INTESTINAL TRACT

	Number of Cases	
Esophagus	2	
Stomach	3	
Jejunum	1	
Cecum	1	
Large intestine	3	
Total	10	(10%)
Rectum (extension)	9	
Total	19	(19%)

The suprarenals, pancreas, diaphragm and pericardium were invaded once, but in all these cases metastases elsewhere were present.

Ocular involvement was present once. Such metastases are rare and either invade the choroid directly or extend to the eye from the involved bony structures around it. Exophthalmos usually occurs. Greenwood[34], in 1902, was the first to report a case of cancer of the prostate metastasizing to the choroid.

Metastatic nodules to the penis are rare (Adenot[35], Wolff[17], Tailhefer[36], Clado[37]). They have only been noted in cases of generalized carcinomatosis. They may appear directly under the skin of the shaft of the penis or within the corpora cavernosa. In this series the corpora cavernosa were invaded once.

Nodules beneath the skin occurred once. They are usually multiple, firm, move freely, and always associated with generalized carcinosis. Bumpus[25] reported one in his series.

Metastasis from carcinoma of the prostate to the central nervous system and spinal dura not infrequently occurs. Of the two, the latter is more commonly involved. In this series, extradural metastases were present three times, and the involvement of the brain (pia) without spinal dura metastasis occurred once. (The spinal cord and brain were not investigated at necropsy in many cases.) Two of the three cases showing extension to the spinal cord had complete paralysis of the lower extremities; in the third, partial paraplegia was present. In two cases the retroperitoneal glands were involved, and associated with one of these cases there was a generalized carcinosis. In the other case, the glandular extension was unique. The third case showed no adenopathy, no seminal vesicular involvement, but a small metastatic nodule in the lung. This unquestionably was embolic in nature. The only case in this group showing metastatic tumor in the central nervous system also showed diffuse carcinomatosis with a large tumor mass in the choroid and involvement of the frontal bone.

In twenty-three cases of central nervous system and spinal dura metastasis from carcinoma reported by Winkelman and Eckel[38], the prostate was the primary source of the malignancy in five cases. In one of these the paraplegia was apoplectiform in onset and was found to be due to occlusion of the blood vessel by the tumor mass rather than by direct pressure on the cord itself. Spiller[39] reported two cases of spinal cord metastasis with sudden paraplegia, one due to carcinoma of the prostate and the other to carcinoma of the stomach. Clinically, one infrequently sees a patient with symptoms from a cord lesion, due to metastasis from prostatic cancer, appearing as the initial symptom. In 200 clinical cases of malignant prostate at the Massachusetts General Hospital this occurred twice. Both patients had paraplegia of the lower extremities, the onset of one appearing more gradually than the other. Similar cases are reported by Thompson[40] and Bumpus[25]. Complete vesical retention was present in both. Operation for cord tumor was performed on one.

In 1000 clinical cases, Bumpus[25] cites spinal cord metastasis in eleven. Several of these had no urinary symptoms. It is surprising indeed that, with the incidence of vertebral metastases as high as it is, spinal dura extension is not greater. Unquestionably, in advanced cases, vague, indefinite symptoms are present which are often overlooked, and necropsies are often incomplete.

In twenty-nine of the fifty-one cases showing ureteral and pelvic pathology, the ureters and pelves were diseased and contained pus. (See table 6.) Eleven of these had bilateral suppurative pyelonephritis. In one case the pyelonephritis was localized to one kidney and a perinephritic abscess was associated with it. In three cases there was bilateral ureterectasis and pyelectasis with infection on only one side. It it difficult to explain the findings in a single case in this group in which infection was found in the ureter and not in the pelvis, and in another case in which the opposite was true. The cause for the ureterectasis and pyelectasis in most instances was due to carcinomatous extension around the walls of the lower portion of the ureters (see figure 5), rather than to infection, for the seminal vesicles were involved forty-two

times in this group. Pelvic and ureteral dilatation without involvement of the seminal vesicles was present in only nine cases. Mechanism of dilatation here could best be explained by associated benign hypertrophy or infection or both. In one case in which no mention was made of vesicular extension, unilateral dilatation and infection of the pelvis and ureter were present. Although ureteral and pelvic dilatation are more apt to be present in cases in which extension to the vesicles has taken place, nevertheless, in fifteen cases of this entire series, in which the vesicles were invaded, no ureteral or pelvic dilatation was present. By the same token, although upper tract dilatation is usually associated with vesicular involvement, yet as previously stated, there were nine cases in this group with upper tract dilatation and no visible involvement. It is significant that in only one instance in this series was the ureter so completely obstructed as to produce a hydronephrotic atrophy of the kidney. Renal calculus was present in only one case, and vesical calculi in two.

TABLE 6

INCIDENCE OF RENAL AND URETERAL PATHOLOGY

Total number of cases showing renal and ureteral pathology	52
Bilateral pyelectasis and ureterectasis and infection	29
Bilateral suppurative pyelonephritis	11
Unilateral suppurative pyelonephritis and perinephric abscess	1
Bilateral pyelectasis and ureterectasis with infection on one side	3
Ureteral infection without renal infection	1
Renal infection without ureteral infection	1
Renal calculus	1
Pyelectasis and ureterectasis with involvement of vesicles	42
Pyelectasis and ureterectasis without involvement of vesicles	9

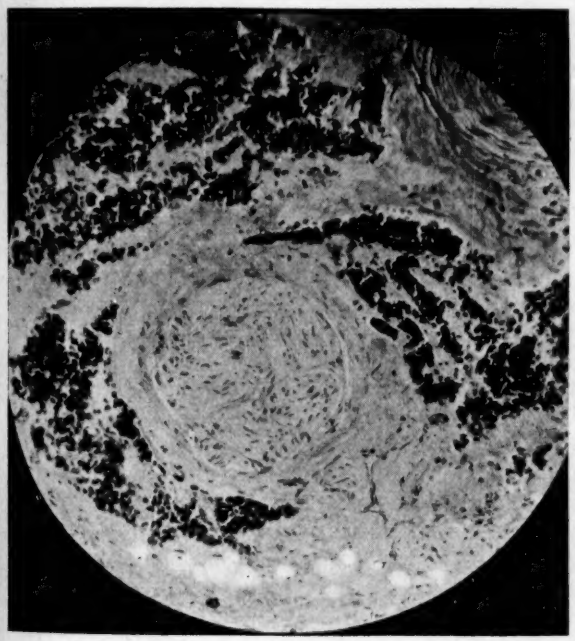

FIGURE 5. Invasion around nerve sheath in wall of the ureter. (Low power.)

Suppurative bilateral pyelonephritis with small cortical abscesses was noted in a great many cases of this group. Metastasis to the kidneys occurred three times, and once to the ureter. In all four instances, glandular, osseous or visceral metastases were associated with it. In no instance was there an isolated renal or ureteral metastasis. In all the cases, and also in some of the literature (Giordano and Bumpus[41], Motz and Majewski[16], Wolff[17], Carson[42]) the nodules were small. The pathway of dissemination of the neoplastic cells may be lymphatic or vascular in origin, or both. Direct extension may be a factor in the invasion of the lower third of the ureter.

The entire retroperitoneal chain of glands was involved in three of the cases showing renal metastasis. The glands around the renal pedicle were also invaded. In one instance a tumor embolus was seen microscopically in one of the branches of the renal vein. On two occasions, both kidneys showed metastatic nodules, and in the other the process was unilateral. The renal nodules were usually multiple.

The autopsy protocols are lacking in data on the occurrence of osseous metastases. (See table 7.) In only twenty-one cases is it mentioned.

TABLE 7

OSSEOUS METASTASIS

(Metastasis to bone recorded in only 21 cases)

	Number of Cases
Vertebra	20
Pelvis	13
Ribs	10
Femur	3
Skull	2
Clavicle	1
Pubis	1

Of these the spine was involved twenty times, the pelvic bones, thirteen, the femur three, ribs ten, skull twice and pubis and clavicle once each. There was no instance of an isolated osseous metastasis, as cited by Blumer[43], without involvement of the spine or pelvic bones. Likewise, in every case showing bone extension, the retroperitoneal lymph nodes were also enlarged.

A few cases of pathologic fractures of the femur were noted, but in each case the spine and pelvis also showed neoplastic extension. Pathologic fractures, contrary to belief, are not uncommon and frequently are reported (Wes-

son[44], Silcock[45], O'Neil[46]). It is surprising that they do not occur more often, considering the frequency with which the femora are involved. With the exception of the ribs and ofttimes the clavicle, spontaneous fractures of other parts of the skeletal system are uncommon. In this series only a few pathologic fractures of the ribs and one of the clavicle were noted. Infrequently, pain in the chest (Brown[47]) and pain in the shoulder girdle may be due to os-

FIGURE 6. Pathologic fracture of the neck of the femur. (Case of Dr. R. F. O'Neil.)

seous metastases to the ribs and shoulder girdle, respectively, from cancer of the prostate. Fracture of the ramus of the pubis has been observed (Wesson[44], Chute[48]), but not one case occurred in this series. They are exceedingly rare. Diffuse osseous carcinosis is usually seen very late in the disease. It was present in about seven cases, but is not uncommon (Putti and Faldini[49], Tartagli and Soli[50], Castiglioni[51], Roccia[52], Thiele[53], Sproul[54], Silcock[45], Bergmark[55] O'Crowley et al.[56]). Although local symptoms may not be present in moderately advanced cases, yet in carcinosis they are usually present. The case of Bergmark[55], with diffuse osseous metastases and local symptoms, is the exception that proves the rule.

The low incidence of bone metastases in this series can be attributed to many factors, chief of which was the failure of the pathologist to record the fact. In many cases, however, the microscopic record on a section of bone is missing. Clinically the incidence of bone invasion, as manifested roentgenographically, is usually much higher than that found at the postmortem table. In 200 cases of prostatic cancer at the Massachusetts General Hospital, x-rays of the spine and pelvis were taken 138 times; fifty-one, or about thirty-seven per cent, of these showed demonstrable metastasis to the spine and fifty-four, or about thirty-nine per cent, had metastases to the pelvic bones. Roentgenograms of the femora were taken 127 times; of these twenty-six, or twenty per cent, showed cancer involvement. Bumpus[25] tabulated the findings on 539 patients with carcinoma of the prostate on whom x-ray examinations of the bones were done. Metastases to the pelvic zones occurred 123 times, the spine 107, femur sixteen, and ribs ten times. Barringer[57] cites forty-six cases, forty-four of whom had x-ray of the spine, pelvis and long bones taken. Thirty-nine of these, eighty-nine per cent, showed invasion of the lumbar spine, pelvis and upper end of the femora. Necropsies as well as roentgenograms show a high ratio of osseous metastasis from prostatic cancer. Blumer[43], in forty-three necropsies, found the pelvis involved twenty-six times, vertebrae thirty-five times, ribs thirty, femur twenty-five, skull eighteen, sternum sixteen and humerus fifteen times. One cannot help but be impressed by the increased incidence of extension to the skull, sternum and humerus in this series. The figures of Carlier and Davrinche[58], in necropsies, are equally impressive. The vertebrae were involved seventeen times, pelvis thirteen, femur eleven, ribs eight, sternum eight, skull, humerus and scapula six times each, tibia three, clavicle two and fibula, radius and ulna once each. In 205 autopsies, Ferguson[8] found bone metastasis in thirty per cent.

The type of osseous metastases, i.e., whether osteoplastic or osteoclastic, was seldom mentioned. In the cases in which roentgenograms of the spine, pelvis and femora were taken before death, the proliferative type of bony reaction was noted in all. A case, such as described by Simpson[59], of diffuse vertebral metastasis, in which the osseous reaction was neither osteoplastic nor osteoclastic, and which did not show by x-ray, was never noted. They are extremely rare. That osteoplastic metastasis gives rise to more pain than osteoclastic metastasis (Waters and Pierson[60]) has never been proved. It seems more logical to assume that the intensity of pain is more closely allied with the location of the metastases and the extent of posterior root involvement rather than with the type of bony reaction. Since pain is a subjective symptom, the individual factor cannot be cast aside. In cases in which microscopic sections of the bone involved were present, it was observed that in almost all of them the structure and characteristics of the primary growth were retained (Phemister[61]). (This applies only to the cases reviewed at the Massachusetts General Hospital.)

It was not disappointing to discover that no clue or suggestions could be found concerning the pathway of dissemination to bone. It is hardly to be expected, for in all probability, all the present recognized routes were involved, in such advanced cases (perineurial, vascular, intraspinous [Roberts[62]], lymphatic). Until our knowledge of lymphatics of bone and bone marrow (Kolodny[63], Piney[64], von Recklinghausen[65]) is increased, the pathway of dissemination to bone from carcinoma of the prostate will be obscure.

The authors wish to thank Doctors Tracy B. Mallory, Kenneth Mallory, Frederic Parker and Shields Warren for the material which made this article possible.

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THE VALUE OF ICTERIC INDICES AND PLASMA LIPIDS IN THE DIAGNOSIS OF JAUNDICE*

BY EARL R. LEHNHERR, M.D.†

INTRODUCTION: Jaundice is the clinical observation associated with the presence of bile pigments in the skin, sclerae, or mucous membranes. Related terms have been used to describe the presence of bile pigments in the blood stream occurring without clinical evidence of jaundice. This latter condition is often called a *latent jaundice* or a *subicteric condition*. Thus, the definition of jaundice automatically eliminates the yellowish color due to carotinemia or picric acid poisoning.

There are two papers on jaundice which should be read by every student of medicine. The first was written by McNee[1] in 1923 and gives an excellent review of the previous experimental work. Rich[2], in 1930, incorporated the interval data and offered a different classification based largely upon the van den Bergh test.

McNee classified jaundice as obstructive, hepatic, toxic and infective, or hemolytic in origin. He designated as latent jaundice those cases with bilirubinemia, as shown by the van den Bergh test, without clinical jaundice.

Rich based his classification upon a detailed consideration of the probable mechanism for the formation and the excretion of the bile pigments. The retention type is due to an upset in the balance between the formation and the excretion of the pigment. The regurgitation type is due to the presence of bile pigments in the blood stream which have once passed through the parenchymal cells into the biliary canaliculi.

Either of these classifications is satisfactory for the pigeonholing of diagnoses, but neither attempts to differentiate jaundice requiring surgical intervention and that type due to liver

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damage which is primarily a medical problem or emergency. It is true that this information will follow a correct diagnosis of the causative factor for the jaundice, but everyone is familiar with certain types of jaundice which have been given to the surgeon when medical treatment was indicated, or else withheld from the surgeon when only operative treatment could be of help. That group with painless jaundice is composed of the patients most often wrongly diagnosed and whose lives are jeopardized.

MEANING OF JAUNDICE: Experimental work points toward hemoglobin as being the precursor of the bile pigments. Hemoglobin is split into hematin and globin. Hematin is in turn broken down into hemosiderin and hematoporphyrin. The latter compound is iron-free and is isomeric with bilirubin which is probably formed in the cells of the reticulo-endothelial system. Bilirubin may be oxidized to other pigments such as biliflavin (yellow), bilixanthin (yellow), bilicyanin (blue or purple), bilifuscin (green), biliverdin (green), bilihumin (dirty blackish), biliprasin (green), et cetera[3]. Some of these pigments may be formed previous to excretion as is seen clinically in subdural hematomas, pleural exudates, hematomas, lung infarcts, purpuric spots, and ordinary bruises. Some pigments may be formed during the process of excretion, and many pigments are formed after the excretion of bilirubin into the biliary system. These changes into pigments, originating from bilirubin, are associated with stasis of the pigment. Thus jaundice may be due to the presence of bilirubin in the plasma, to the derivatives of bilirubin in the plasma, or to the retention of formed bile (derivatives of bilirubin, free cholesterol, phospholipid, bile salts, mucin, inorganic salts, et cetera) in the plasma. I have tried to think of these theoretical possibilities as entities and have associated them with the terms bilirubinemia, biliemia, and cholemia. The latter term may be confusing with the retention of bile salts, likewise termed a cholemia, and perhaps a more suitable term may be suggested. An overproduction of bilirubin too great for the usual functioning liver (rare) or a reduction in bilirubin excretion will be associated with a bilirubinemia. If this upset continues, a portion of the bilirubin will be changed to other pigments. This condition will no longer be a bilirubinemia, but a biliemia. An obstruction to the normal bile flow with the accumulation of whole bile in the blood stream is also a biliemia when referring to the pigment stasis, but it is more correctly termed a cholemia because there is a retention of all biliary substances. This term should be restricted to obstructive jaundice.

THE MEASUREMENT OF JAUNDICE: Inspection is the easiest and most commonly used method for the measurement of jaundice. It is commonly looked for, and compared from day to day, in the skin, sclerae, and mucous membranes. The character of the color may vary from a golden yellow or greenish yellow to a dirty yellow color. The icterus may be masked in the skin of colored people, but it is readily distinguished in the sclerae or in the frenulum under the tongue. The tissue may be stained by the pigments so that the patient may be jaundiced after the bilirubin has disappeared from the plasma. However, there is bound to be some pigment, if not bilirubin, in the plasma so long as bile pigment is being mobilized from this stained tissue.

The various colors observed are sometimes associated with certain clinical conditions, the golden yellow and greenish jaundices often being interpreted as being due to intrahepatic or obstructive jaundices respectively.

The laboratory methods measure the bilirubin content by the van den Bergh test[4], or estimate the color of the plasma (or serum or acetone extract) against standard potassium dichromate solutions[5, 7, 8]. The estimation of the bilirubin content will represent accurately the amount of jaundice in bilirubinemia, but it will have no relationship to the bile pigments in biliemia. The icteric index is meant to measure the relative amount of coloring matter in the plasma or serum. It will, in bilirubinemia, give values closely related to the bilirubin content, but in biliemia (or cholemia) its values will be higher. The color of the plasma is usually yellow in character and may match the various concentrations of dichromate. However, biliverdin or bilihumin may be present and the color cannot be matched against dichromate standards. Neither of these methods, though accurately carried out, represents the true pigment content of the plasma. There are quantitative methods for bile pigment which attempt to eliminate these discrepancies[6].

The use of sera in the determination of icteric indices is routine in many clinical laboratories. The contraction of the fibrin during the formation of the clot serves to filter out many of the derivatives of bilirubin and the resulting sera will not represent the true icteric index of the circulating plasma. The use of heparin and the hemolysis of red cells also introduce errors in the reading of the icteric indices. Plasmas containing large quantities of bilirubin may give a relatively high icteric index if the plasma is undiluted. This is not important, because with a bilirubin concentration of 8 to 15 milligrams per cent there is a biliemia of some magnitude. There is another practise indulged in by some laboratories in attempting to determine accurate icteric indices. The technician realizes that the color of the dichromate standard and the plasma are not comparable in diffuse light, but are readily compared in stronger light which tends to eliminate the deeper colors in

the plasma. Likewise, the use of small tubes for the standards and the unknown plasma tends to diminish the color introduced by some of the bile pigments. These practices should be condemned if a true icteric index is desired.

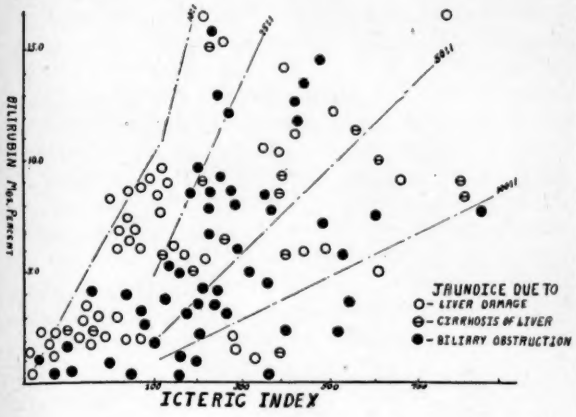

CHART 1.

In chart 1 is shown the relationship between the icteric indices and the bilirubin content of plasmas from jaundiced patients. These data have been associated with clinical jaundice due to liver damage, cirrhosis of the liver, and to obstruction of the normal bile flow. The diagonal lines serve to group the icteric indices and the bilirubin values in their ratios, such as 10:1, 25:1, 50:1, and 100:1. This chart shows that the ratios found in jaundice due to liver damage are grouped near the ratios 10:1 and 20:1. It also shows the possibility of large discrepancies in either type of jaundice. Therefore, a comparison of the icteric index and the bilirubin value in a given case of jaundice will not give absolute information as to the etiology of the type of jaundice. This chart emphasizes the difference meant by the terms bilirubinemia and biliemia.

LIVER FUNCTION TESTS: Any attempt to differentiate jaundice needing surgical or medical treatment necessitates the use of a test which will give information as to whether the jaundice is due to injury of the parenchymal cells or to obstruction of the normal flow of bile. Likewise, it should be able to tell the margin of safety between a reasonably good functional activity and serious or even fatal breakdown of the liver parenchyma.

The functions of the liver are many and widely diversified[9]. It is the largest gland in the body and is composed of three distinct types of cells, the parenchymal, biliary, and Kupffer cells. The parenchymal cells are known to be closely related to the metabolism of carbohydrates, lipids, proteins, water and electrolytes[9, 10, 11, 12, 13]. The better known function tests for these cells consider variations in the galactose tolerance, plasma protein and fibrin content, non-protein nitrogen allocation, lactic acid in the blood, and the red cell volume[14, 15, 16]. A less generally known function, and poorly understood, is its relation to water metabolism[12, 17]. The parenchymal cells probably have nothing to do with the formation of bilirubin. The intact biliary cells and capillaries are necessary for the excretion of the formed bile. Thus a retention of bile pigments by the liver may be due to a destruction of the parenchymal or biliary cells, or to a change in the patency of the biliary canaliculi. The Kupffer cells are related to pigment metabolism. Certain dye tests have been recommended for the determination of the liver function[18]. These are introduced intravenously and are excreted by a normal liver. Their retention in the blood stream may be due to either parenchymal damage or to biliary obstruction and need not necessarily indicate parenchymal cell damage as the cause of the jaundice.

Bilirubin excretion tests have been introduced and are claimed to be more satisfactory than the dye tests[19,20]. It, too, depends upon the combined functional activity of the parenchymal and biliary cells. Presumably it would be impossible to differentiate a jaundice due to liver damage or that due to a complete obstructive lesion of any duration.

It is not possible to perform a single test for the liver functions which will give a value absolute in its interpretation. One writer[21] suggests a minimum of laboratory investigation in the study of hepatic function to include, (1), sugar tolerance test, (2), galactose tolerance test, (3), determination of the amino acids in the blood and urine, (4), determination of the bilirubin in the blood, (5), determination of the biliary acids in the urine, (6), determination of the total bile pigments in the blood and urine, (7), determination of the cholesterol in the blood, (8), roentgen study of the gastrointestinal tract and cholecystography, and (9), esophagoscopy for the detection of varices.

PLASMA LIPIDS IN JAUNDICE: The liver has been shown to be related to the formation of lipids and the preliminary desaturation of the fatty acids preparatory to their metabolism[10, 11]. When we began this series three years ago there was little accord as to the interpretation of the plasma lipids and cholesterol values. This accounted for the statement of Peters and Van Slyke[22] that "the results of blood lipoid studies in diseases of the liver have been disappointingly conflicting." During the interim several papers have been published giving what we believe to be a correct interpretation of the plasma cholesterol related to the functional activity of the liver[23, 24].

The plasma lipids are composed of several different types of compounds. These are all basically fatty acid derivatives, combinations of fatty acids, or compounds capable of combining with fatty acids. They may be grouped according to their physiological activity. This activ-

TABLE 1

ICTERIC INDEX, BILIRUBIN, HEMATOCRIT READING, AND PLASMA LIPIDS IN JAUNDICE

Etiology of Jaundice	Cholesterol Total*	Number Cases	Determinations	Icteric Index	Bilirubin*	Red Cells Per Cent (Hematocrit)	Cholesterol* Total	Ester	Free	Per Cent Free	Fatty Acids*	Phospholipids*	Total lipids*
Liver Damage	Less than	3	5	250	8.2	41	37	1	36	97	153	.....	.....
Cirrhosis of Liver	50 mgs.	0	0	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Biliary Obstruction		0	0	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Liver Damage	51-100	8	20	65	4.6	38	84	31	55	65	280	158	370
Cirrhosis of Liver		2	2	650	11.0	48	96	17	88	92	273	.....	369
Biliary Obstruction		0	0	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Liver Damage	101-150	10	28	186	6.5	36	122	38	85	70	445	223	564
Cirrhosis of Liver		8	14	70	3.7	37	130	60	69	53	416	221	534
Biliary Obstruction		0	0	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Liver Damage	151-200	0	0	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Cirrhosis of Liver		13	19	410	8.3	39	183	65	119	65	653	267	844
Biliary Obstruction		0	0	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Liver Damage	201-300	0	0	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Cirrhosis of Liver		8	12	415	5.0	44	235	94	149	64	421	234	634
Biliary Obstruction		10	16	272	4.3	32	264	104	150	58	685	260	944
Liver Damage	301-400	0	0	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Cirrhosis of Liver		0	0	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Biliary Obstruction		7	14	235	6.4	32	350	54	286	82	795	354	1132
Liver Damage	400-	0	0	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Cirrhosis of Liver		0	0	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Biliary Obstruction		8	13	312	7.0	39	532	132	408	76	1520	389	1720

*Recorded in milligrams per cent in oxalate plasma.

ity is based upon the presence of desaturated fatty acids in the combination. The most active, i.e., the most easily oxidized, are the phospholipids. Cholesterol combinations with unsaturated fatty acids, neutral fat, sphingomyelin and cerebrosides (not occurring in the plasma), cholesterol combinations with saturated fatty acids, and free cholesterol follow in this gradient.

There is one physiological invariable which is described as being associated with all living tissue. This is the élément constant by which is understood the minimal amount of liquid in tissue compatible with life. We have tried to transfer this conception to the blood stream, it being living tissue necessitating a certain amount and quality of lipid during the active life of the organism. This low lipid value might be limited to an amount compatible with life, lower values occurring during an irreparable or serious upset in body economy. The blood stream élément constant might be only a mirror of the metabolic changes taking place in other tissue and a low plasma lipid being the result of a breakdown in equilibrium elsewhere in the body with reduction of the lipid content to the theoretical élément constant. If the liver could be hooked up with lipid mobilization, an approach of the lipid values in jaundice to or below the élément constant would be encouraging, because with obstruction in the biliary tract there is an accumulation of free cholesterol and other lipids which are normally excreted in the bile. Likewise, the lipids retained in biliary obstruction probably differ from the lipids normally present in the plasma, because they are products which the body excretes in a similar manner as it excretes urea, uric acid, and creatinine through the kidney. A retention of those lipids should be mirrored in the character of the plasma lipid similarly as the non-protein nitrogen allocation is changed in kidney insufficiency. Thus, if the liver is associated with lipid mobilization and excretion, there should be diametric plasma findings in jaundice due to parenchymal cell damage and that due to biliary tract obstruction.

In table 1 is shown the plasma findings from approximately eighty cases of jaundice. The etiological diagnosis has been confirmed by operation, postmortem examination, or by successful medical treatment. They have been grouped in three columns, namely, jaundice due to parenchymal cell damage, jaundice associated with cirrhosis of the liver, and jaundice due to the obstruction of the bile flow. All of these groups are important for correct diagnosis from the surgical and the medical point of view.

The explanation of these groups should not need elaboration. The jaundice due to parenchymal cell damage and that due to obstruction of the bile flow should be clear. Two cases of hemolytic jaundice (familial type) have been grouped with jaundice due to parenchymal cell damage. This would agree with those workers who believe that the normal functioning liver is able to take care of an excessive bilirubin formation[2, 25]. The jaundice associated with cirrhosis of the liver is not clear. In this group has been placed cases of toxic jaundice, infectious jaundice, catarrhal jaundice, acute hepatitis, alcoholic cirrhosis, et cetera. They were all associated with some process superimposed upon a previously injured liver. The nature of the superimposed process is not known, but it may be related to the sensitivity of the parenchymal cells to a poison or toxin, lack of calcium, or a high protein diet. In 80 per cent of the cases the plasma chemistry was similar to that found in jaundice due to liver damage. These cases may well represent an early picture of liver damage whereby the amount of plasma lipid is normal, or actually increased through the absorption of cholesterol from the gastro-intestinal tract after the liver function has been diminished through damage, but their character is changed because of the diminished liver excretion.

Reference to table 1 shows that the jaundice due to liver damage (parenchymal cell injury) is associated with diminished plasma lipids, low free and ester cholesterol, and an increase in the free-cholesterol:ester-cholesterol ratio. Jaundice associated with cirrhosis of the liver shows low, normal, or high lipid values with an increase in the free-cholesterol:ester-cholesterol ratio. The obstructive jaundices are associated with increased plasma lipids, increased free-cholesterol, and increased free-cholesterol:ester-cholesterol ratios. The icteric indices, bilirubin content, and hematocrit findings were similar in each group.

There is one significant finding common in each group of the jaundiced patients, namely an increased free-cholesterol:ester-cholesterol ratio. This phenomenon has been termed "ester-Kurz" by Thannhauser[13, 26]. It is interpreted as being due to a loss of the esterifying activity of the liver cells. Thus, the determination of the free and ester-cholesterol values will give information as to the type of the jaundice (i.e., due to parenchymal cell damage or biliary obstruction) and a fair picture of the severity of the liver damage secondary to the obstruction of the bile flow. This secondary liver pathology is not considered in the etiology of the jaundice. It is merely a process found in varying amounts in obstructive jaundice. It is important for the treatment and prognosis of the surgical patient before and after operation.

The diagnosis of jaundice by means of plasma lipids is not so easy as the table might tend to show. Many determinations have been omitted from the tables in order to show the effect of an actual lesion. Early, intermittent, incomplete, or subsiding obstruction jaundice may have a

normal plasma lipid with definite biliemia, because of the continued mobilization of fixed bile pigments from the stained tissue. A careful history and physical examination are requisite to the correct interpretation of the plasma chemistry.

Summary

(1) Jaundice is not a simple bilirubinemia. It may be due to bilirubin or to any of its derivatives in the plasma; and its clinical appearance in the skin, sclerae, and mucous membranes will vary accordingly.

(2) Neither the bilirubin nor icteric index measures the total bile pigment in the plasma. The plasma may contain bilirubin without clinical evidence of jaundice, or contain other bile pigments without bilirubin. This latter condition is commonly found in subsiding jaundice and is due to the mobilization of bile pigments temporarily fixed in the stained tissues.

(3) The ratios of the icteric index and bilirubin content approach 10:1 as the color of the plasma is due primarily to bilirubin.

(4) Jaundice due to parenchymal cell damage tends to have low plasma lipids and increased free-cholesterol:ester-cholesterol ratios. This included jaundice associated with cirrhosis of the liver and familial hemolytic jaundice.

(5) Jaundice due to biliary obstruction tends to have increased plasma lipids with increased free-cholesterol:ester-cholesterol ratios.

(6) Increased free-cholesterol:ester-cholesterol ratios are presumably an index of the parenchymal cell damage in all types of jaundice.

(7) The plasma lipids in jaundice can be explained if the undamaged liver is necessary for the mobilization of lipids, the esterification of the free cholesterol, and the excretion of the lipids from the body.

(8) A careful history and physical examination of the jaundiced patient are necessary for an interpretation of the etiological process present in the liver.

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AN UNUSUAL CASE OF TRAUMATIC HERNIA

BY DUNLAP P. PENHALLOW, M.D.*

ALTHOUGH it is generally conceded that herniae in general are not due to trauma but are usually caused by the gradual protrusion of some organ, through points of anatomical weakness, there are instances in which trauma apparently plays an important part in the production of a hernia, as for instance in the case of certain types of diaphragmatic hernia which appear after an injury; and also the postoperative, or postincisional types.

A search of the literature fails to reveal a hernia similar to the one to be described, the nearest approach being those herniae which occur in the linea transversalis or so-called Spigelian herniae. The one reported herewith, however, showed some protrusion of the intestine into the opening simulating a Richter's hernia, although the protrusion of the intestine was very

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slight. It is felt to be a true traumatic ventral hernia and was probably the result of an injury to the muscles of the abdominal wall.

L. W. S., aged fifty-five, was seen in January, 1932. He stated that about twenty years ago, while playing baseball, he was struck on the abdomen by a

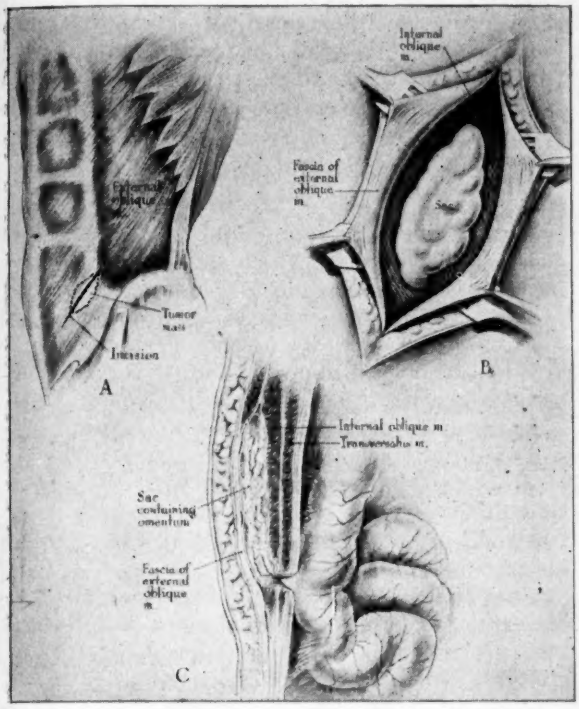

FIG. A. Location of tumor mass should be slightly lateral to edge of rectus muscle.

hit ball. He suffered intense pain at the time of injury but this pain gradually wore off and was replaced by a feeling of discomfort at the site of injury, which discomfort was intensified if he sneezed or coughed. At those times the pain would be intense and in order to get relief it was necessary for him to press on his abdomen at the site of the pain. Later he began to notice a small swelling at the site of injury, and this lump has been gradually increasing in size. At times the mass is larger than at other times, but it never entirely disappears. He also states that when the mass is larger there is a greater sense of discomfort and pain often associated with nausea and vomiting.

EXAMINATION: Well developed and nourished male, good musculature. General physical examination essentially negative save as follows: *Abdomen:* On the right side is a well-healed appendectomy scar. On the left abdomen, two inches above the inguinal canal and just to the outer side of the left rectus muscle is a definite tumor mass, most noticeable when the patient stands, strains and coughs. (Fig. A.) In the recumbent position the mass appears to reduce and a small defect can be felt in the abdominal wall. There is a definite impulse on cough. The mass is situated well above the inguinal canal. A gastro-intestinal series was done, but no abnormalities of the gastro-intestinal tract were found.

DIAGNOSIS: Traumatic Ventral Hernia.

Operation was advised and accepted.

OPERATION: Elliptical incision was made over the tumor mass in the left midquadrant and carried through the fascia and muscle planes at this level. When the fascia of the external oblique muscle was split an irregularly elliptical flattened out sac was found lying on the internal oblique muscle, and when this sac was opened it was found to contain omentum. There was a small neck leading to an orifice in the muscle which orifice was about 1 cm. in diameter, perfectly circular, and tightly gripping the base of the protruding mass of omental tissue. (Figs. B & C.) The fascial planes and muscles were carefully laid open exposing the channel of the hernia. The omentum protruding through was tied, excised and removed, and loops of small intestines adherent to the anterior abdominal and lateral abdominal wall resulting from long-continued irritation were freed from these attachments and the denuded surface carefully covered with peritoneum. The incision was closed by careful muscle approximation and overlapping of fascial planes, obliterating the canal and the hernia. Sutures in the muscle and fascia were No. 2 chromic and in the skin continuous linen. Following the operation the patient made an uneventful convalescence and the symptoms of which he had complained entirely disappeared.

CONCLUSION: I believe this hernia to be a rather unique type and also that it can be classified as a true traumatic hernia, the result of a localized weakening or rupture of the muscles of the abdomen by the blow received by the struck ball.

REPORT OF CASE OF TORSION OF THE SPERMATIC CORD IN A BABY TEN MONTHS OLD

BY FRANCIS F. HENDERSON, M.D.,* AND PHILIP S. FOISIE, M.D.*

THE occurrence of this comparatively rare condition in such a young patient seems to warrant its being reported.

The case in question is one of a baby ten months old, who had previously been noted to have a hydrocele on the right side. The parents had been advised to have this condition repaired, but they had refused and the hydrocele had been tapped with temporary relief. About two months following this, the mother noticed that the patient seemed to be in pain, and cried almost constantly. She also noticed a swelling in the left inguinal region. She applied hot cloths and gave the patient various home remedies which apparently did not relieve the condition. About twenty-four hours after she first noticed that the patient was having trouble, she consulted the senior author. At that time, the baby appeared to be in considerable pain and it was found that the left testicle was not in the scrotum and there was a hard, tender mass in the region of the left external ring. There were no abdominal symptoms, and the baby's bowels were said to have moved freely that morning. The patient was immediately taken to the hospital, and under ether

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anesthesia, an incision was made over the external ring. The skin and subcutaneous tissues were extremely edematous. Coming down to the external ring, the testicle was found pulled firmly up against the fascia of the external oblique. The fascia was split parallel to its fibres a short distance, revealing a spermatic cord which was engorged and twisted counter-clockwise one and one-half times. The testicle and epididymis were black. When the torsion was corrected, hot cloths were applied to the testicle and epididymis for some time, but no change was visible in their condition. It was obvious that the blood supply was permanently cut off, and they were amputated. After removal of the testicle, a small direct hernia was noted and the abdominal wall was firmly sutured for the repair of this condition. The patient made an uneventful convalescence.

The rarity of this condition in such a young patient was evident when we found a report by Taylor of only one other case in a child near this age. In his case, symptoms were first noticed four hours after birth, but the patient was not operated on until two days later, at which time the testicle and epididymis had to be removed. To date approximately one hundred and twenty-five cases have been reported; about 75 per cent of these occur between the ages of fifteen and twenty-five years, and the oldest recorded is at the age of sixty. This condition is definitely one of young men near twenty. In about 50 per cent of these cases, the condition was associated with an undescended testicle. A great many of the surgeons reporting cases made the observation that there was an abnormal attachment of a long mesorchium which enabled the testis to rotate.

A large variety of inciting causes has been given; the most common of these are heavy lifting, straining, and trauma. There are several cases on record, however, of the condition coming on during sleep, so that evidently the precipitating cause may be varied or unknown.

The condition is chiefly confused with strangulated hernia and acute epididymitis. Absence of abdominal symptoms and absence of previous venereal infection are therefore important points in the differential diagnosis.

There are apparently some cases which have a small degree of torsion and which right themselves, thereby causing temporary acute symptoms which subside. It is said that there have been patients who have had the condition frequently and have learned to correct it themselves by manual manipulation. If the torsion is extensive and untreated, of course gangrene develops but in some untreated, mild cases (about 14 per cent) where the torsion is not extensive enough to cause gangrene, atrophy of the testicle develops.

SUMMARY: Torsion of the spermatic cord resulting in gangrene of the testis is a condition occurring chiefly in young adults. In this paper a case is reported in which torsion occurred in an infant ten months old.

The predisposing cause is apparently an abnormal attachment of the mesorchium.

Where the condition is found, it is commonly associated with other abnormalities of these organs, such as undescended testis, inguinal hernia, and hydrocele.

The precipitating cause has been thought to be trauma of some sort, but such trauma is not essential as some cases have occurred during sleep.

The condition may be distinguished from strangulated hernia by the absence of intestinal obstruction.

In mild cases the condition sometimes corrects itself or results in atrophy, rather than gangrene.

Early diagnosis and early operation are essential in order to save the testicle.

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MORTALITY RATES

Telegraphic returns from 86 cities, with a total population of thirty-seven millions for the week ending August 25, indicate a mortality rate of 9.3 as against a rate of 9.0 for the corresponding week of last year. The highest rate, (18.1) appears for Memphis, Tenn., and the lowest (3.2) for South Bend, Ind. The highest infant mortality rate (16.5) appears for Atlanta, Ga., and the lowest for Cambridge, Mass, Fall River, Mass., Knoxville, Tenn., Lynn, Mass., New Haven, Conn., Oakland, Calif., Somerville, Mass., South Bend, Ind., Trenton, N. J., Wilmington, Del., and Youngstown, Ohio, which reported no infant mortality.

The annual rate for 86 cities is 11.7 for the thirty-four weeks of 1934, as against a rate of 11.1 for the orresponding period of the previous year.

SUMMARY OF DEATHS AND DEATH RATES (ANNUAL BASIS) FROM AUTOMOBILE ACCIDENTS PER 100,000 ESTIMATED POPULATION FOR 86 CITIES FOR CORRESPONDING PERIODS OF 1934 AND 1933.

	Week ending Aug. 25, 1934	Aug. 26, 1933	First 34 weeks 1934	1933
Total deaths	141	147	5,292	4,800
Death rate	19.6	20.5	21.7	19.7
Deaths due to accidents in city	103	116	4,182	3,780
Death rate	14.4	16.2	17.1	15.5

—*Bureau of the Census.*

THE NEW HAMPSHIRE MEDICAL SOCIETY

PRESIDENT'S ADDRESS*

BY ROBERT J. GRAVES, M.D.†

EVEN the firmest of foundations may be shaken if the applied force is sufficiently great.

When an ancient and honorable society like ours has been fortunate enough to have had one man as its secretary for more than a quarter of a century, his death must needs shake us to the very rock on which we were founded.

No one could have worked with Doctor Sullivan without realizing how much this organization meant to him and what a large part of our structure he was. We shall continue to miss him more and more, personally, professionally, and as a medical group. No matter who is elected his successor, be he ever so able an executive, this society will for a long time to come find itself struggling to reconstruct parts of its edifice that have been shattered through his death.

So far as the memory of any present member goes, we have had but two secretaries, Granville P. Conn and Dennis E. Sullivan. The Society they have so nobly helped to build, and more particularly strengthen, goes on to ever-increasing usefulness. We pause and return thanks that we have been so greatly blessed in our Secretaries.

If this year's presidential address is worthy of any title, I believe it may well be headed by the question "How Far?" How far toward Moscow, toward Rome, toward Berlin, or even how far toward Washington? This question applies not only to political affairs but to almost everything to-day, including medicine. It is truly an age of changing uncertainties.

Sixty-four years ago Dr. Holmes wrote,

"Age lends the graces that are sure to please;
Folks want their doctors moldy like their cheese,"

and four years ago a sister in California wired her sick brother in Boston concerning his physician, "If Dr. —— is over forty, discharge him." Thus medical fashions alter with the times. These changes, however, are elementary and of little significance compared with those which are practically bound to come in the next few years and which are involved so intimately with the socio-economic problems of the day.

Even the doctor who ended his poetical wail with the joyful pean, "Thank God they're having babies in the same old-fashioned way," would have to retract.

Whether this be revolution or evolution, we do not yet know. We hope and believe that there will be continuous increase of medical knowledge as there has been in the past, but there are definite grounds for fear that this progress may be interrupted during the tremendous readjustments that are taking place in what may be called the administration of medical practice.

The near exhaustion of the alphabet for the designation of new functions of government is but one indication of change and confusion. These "alphabetical experiments" while not directly applicable to us as medical men, affect us indirectly in many ways and are of direct concern to us in a few. There has been, to be sure, no R. G. P. A. or M. P. R. A. (Rescue of the General Practitioner Act, or Medico-Public Relations Act) or similar designations for us to tie up with, but we are vitally involved, nevertheless, both in the direct problem of recovery, and in the marked tendency of state and national governments to take over what have been heretofore local or individual functions.

For many centuries medical men have been working under a recognized code which in the main has served well and for the benefit of all concerned. Certainly it is one of the things which have come down to us from antiquity that does not require marked alteration or much modern interpretation. But it does need a few short amendments. Are we going to write these ourselves or be obliged to accept what some socio-political group writes for us?

The health of every individual in a civilized community is a matter of community concern and responsibility. In his accurate way, Dr. Dublin has demonstrated that our vital capital, that is, our health, in prosperous times exceeds our material wealth in the ratio of about five to one. How much greater the ratio is to-day we can merely guess. Thousands to-day count health versus wealth in a ratio of about a hundred to one, and can only ask, "Little man, what now?"

Medical care has been officially declared a necessity of life, and we are its present administrators. Current social and economic trends, however, have tended to accentuate the rôle played by public authorities in this administration.

Last year House Bill 417* was but little more than a name to most of us; now it has become a real factor in the daily professional life of many of our members. Just like other seem-

*Read at the Annual Meeting of the New Hampshire Medical Society at Manchester, May 15, 1934.

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*This bill refers to Welfare Relief and it concerns the Doctors in that under this bill they are now being paid nominal fees for taking care of what were formerly charity patients.

ingly well-conceived efforts involved in the wild scramble to hasten the national and state programs for economic recovery, many snags have been encountered. It is probable that most of these might have been avoided had the time for preparation of the working scheme been longer. Here as in so many other things theory and practice did not always mesh smoothly. At first the administration of the bill was faulty in so many ways that I cannot pause to enumerate them. After five months of trial I found myself in a position similar to that of a bond salesman for a highly reputable house which had widely distributed what seemed to be triple-A securities, only to have them go sour before the first coupons came due. Just at that low point something changed the picture and the machinery began to click.

Cases were being handled better by both investigators and doctors, and checks were coming through with reasonable promptness. The broadside which I was about to dictate remains now only as a memorial of the then despair.

Your coöperation has been splendid and I am grateful. Since the first of this year most of the misunderstandings have been easy to straighten out and it is still quite conceivable that by December 31st many of us will vote 417, what I believe it to be fundamentally, a wise bill and one the major tenets of which should be continued. It has been, however, for many of us, our first real taste of that unpalatable dose, State Medicine. Not that it is really our first dose, since we have had for many years, in fact ever since the State Board of Health really began to function, State Medicine in homeopathic doses, but in ever-increasing potencies. We have accepted this in epidemiology as entirely right and proper. But how far is governmental control over the practice of medicine to be allowed to go?

If we are willing, as we should be, to accept as true Newsholme's postulate that "No sick person can be allowed to lack all that is practicable and really necessary for his skilful and humane treatment, and for his expeditious return to health, or failing this for his comfort while ill," it appears evident that the State must enter the picture, since the existing private agencies cannot fulfil these specifications. Moreover for a large section of the population the cost of sickness is overwhelmingly great.

The rich and the very poor for years received excellent medical care. Those of the great group between have often been deprived of skilful medical attention because they were not paupers nor yet could they afford everything that medicine of the day offered for relief or cure. To be sure they often demanded investigations or treatment which the particular case did not indicate as necessary.

To illustrate this: there is still a widespread idea that an x-ray will furnish the lacking information in every case. The roentgenologist of a large city hospital tells me that over forty per cent of the cases going through his department are entirely unwarranted by any clinical data. The patients or their friends demand x-rays and they have to be taken. To a large extent we have brought this situation upon ourselves. Year by year we have so added to our knowledge of disease and its alleviation that the mass of it is beyond the concept of any one or even any ten men.

The layman is made aware of these advances through articles in magazines and newspapers, by sick friends returning from hospitals cured of strange diseases, and in many other ways. People are apt to believe what they read on matters pertaining to their health. It is said that the first year after the Chicago Gas Company put up its illuminated signs "You can do it better with gas," suicides by gas increased some 200 per cent.

We have never held back knowledge of real value from the public and never will, but sometimes the public demand things that are not for their own good. For instance, "information about girth control is far more important than information about birth control."

Wells, quoting an imaginery DeWindt, says, "It is no good asking people what they want. That is the error of democracy. You have first to think out what they ought to want, if society is to be saved. Then you have to tell them what they want and see that they get it."

This may be a dream of the future. We have to deal largely with the present and recognize the ever-increasing demand for ever-increasing treatment. Just how shall this situation be met, this year and the next and the next?

Time was when the general practitioner, so-called, better the family doctor, held sway and his advice was in the main of a high order and was followed. He knew what the patient needed and saw that he got it. Now with the patient diagnosing his own case, or perhaps as is more often done by the advice of well meaning but poorly informed friends, he needs must go direct to a specialist.

Exactly what *that* is I don't quite know, but at any rate the name is in altogether too constant use and will probably have to serve.

In the old days each doctor had his families and guarded them jealously in sickness and in health. Nowadays the medical man who can count as his alone any half dozen families is certainly the enviable exception. Many families have as many different doctors as there are individual members, and it is partly because of this that there has been an increasing amount of untilled medical ground and the scrub

growth, if you like, of State Medicine has been allowed to spring up.

One example follows. The past few years have witnessed a proper but sadly belated recognition of the importance of prenatal care. Until the state took it over a majority of expectant mothers had little or no skilled supervision during at least six of the nine very important months of the life of a future citizen.

You say that this was the fault of the parents and not of the doctor. Primarily right, but if the doctor had carried on the proper amount of educational work in his community, such a state of affairs would not have existed so largely.

If you will examine the matter carefully, I believe you will find that in most instances such usurpations of medical work by the State have come about because the doctors were either so busy caring for the acutely ill, or so fearful that they would be criticised for drumming up work or for butting in, that an important field has gone uncovered. We can't afford to be dogs in the mangers; we must either do this and similar work, or the people will continue to demand, as they do now, that the State do it.

Frequently a patient will come in for a physical check-up, saying of his regular physician, "He's a fine doctor when you're sick, but he isn't worth a — when you're not," which simply means that his is not a proper medical or social attitude.

The family doctor at his best is the only one who can understand the individual as a whole, sick or well; his environment, his reactions, and his stimuli. No sort of specialist can do this, not even the supertrained psychoanalyst.

But the state, in carrying out its prenatal work or its tuberculosis campaign, or its correction of defects in school children, often goes to homes already adequately covered by a competent medical attendant. The correlation of the various departments, which are necessarily striving to justify their existence, is not thorough and there is a considerable duplication of effort. Not only this but there is a well-founded feeling of irritation on the part of many doctors, that the unsolicited visits of state-employed nurses are unjustifiable because they are trespassing upon the physicians' domains.

It is inevitable if the state takes over more and more medical functions, that the taxpayers, or perhaps more correctly the political heads, will demand an ever-increasing voice in administration of such matters. This will be particularly true with the certain marked increase in hospitalizations. No privately maintained hospital can hope to meet the demands upon it without direct governmental aid, and when this comes about, politics and waste are apt to come in at the same time, to say nothing of much that is medically distinctly distasteful.

How are we going to meet such undesirable possibilities? Not, I hope, as in Russia where every doctor is an employee of the state working on a fixed five-day, eight-hour schedule for a fixed and minimum wage. Not I hope as in one state by doctors becoming the employees of a corporation renting its physicians to its subscribers as a sort of lending library at so much per. Not in many other progress stultifying ways which are being experimented with in various parts of the world and even in our own America.

We are, after all, quasi-public servants, on a reasonably high plane to be sure, but nevertheless servants furnishing a necessity of life. In one sense we are not unlike the various public utilities originally privately owned and operated, which by reason of scientific advance and demand for such services, have at last been brought under state control. We certainly do not want either a holding company or a state commission dictating our professional lives. What are we going to do to prevent it?

In the first place we must sell to the public in every legitimate way the idea of the family doctor for the whole family, making the family the unit and holding its own doctor responsible for it by prevention and prophylaxis first, and then if necessary for the primary diagnosis and treatment of disease should such develop. He must be able to recognize his own limitations and have made available for him consultants really expert in their particular fields.

Our state is pretty well provided with hospitals and other facilities for caring for the sick. Our roads are good and in the main, by virtue of this and the automobile, practically every family in the state is really nearer a doctor in the matter of time than ever before in our history.

Many types of cases can be cared for to better advantage both medically and economically in hospitals than in homes, and it is possible to develop a plan whereby every physician in the state can use some hospital plant to the benefit of both his patient and himself. A greater real welding together of hospitals and doctors, especially those in more isolated locations, is desirable and should be encouraged.

But in order to get anywhere with this idea, there must be brought about a better community spirit between the doctors themselves in each community and the doctors of the state at large.

Some towns in their medical animosities are much like the Balkan kingdoms, ready to fight on the slightest provocation. We have, as doctors, got to get over, what in governments would be called nationalism, and become, as it were, internationally-minded. Another war is surely coming, largely because of this lack of internationalism, and by the same figure, unless we

as doctors get together and meet the changed situation that has arisen both socially and economically, we shall get state and governmental regulation of no pleasurable sort.

We are apt to be declared medico-economically bankrupt and the receiver who will be appointed will not be friendly.

There are some phases of medical practice which obviously can be carried on only by the state. For instance, the important matter of sterilization of feeble-minded and insane persons, both male and female, is properly one which should come under direct governmental regulation.

By authority of law in New Hampshire certain types of individuals are now sterilized for the common good, the operation being performed after definite legal exactions have been met, but on the basis of real state welfare such operations are too few.

Young adults, physically healthy and mentally sadly deficient, are turned loose each year at the period of their most vigorous sexual life and their progeny are all too often predestined to become state charges.

One young feeble-minded girl, released from a state institution because she had reached the age when she could no longer be kept there legally, gave birth to thirteen illegitimate children all of whom became state charges. This is of course an extreme case, but similar histories, on a smaller scale, are being written annually.

Wholly apart from any other argument, the future welfare of our state demands the extension of properly supervised sterilization procedures.

A recent check-up reveals that there are over eleven thousand defectives with respect to teeth, tonsils, vision or hearing among children below high school age, and from the economic group at present unable to provide correctional therapy, either in whole or in part. This means that about sixteen per cent of our school children are handicapped to a greater or less extent in their struggle for existence.

Some well-meaning enthusiast will immediately rise and demand more clinics, but with the already existing agencies 120,000 defects have been corrected in the past five years through the coöperation of the doctors, dentists and school officials; a record for every one concerned to be proud of.

Theoretically we are responsible for the nation's health, practically we have done little but take care of its illnesses.

No community can afford to have poor doctors. Its medical men must receive sufficient income so that they can keep both their mental and physical equipment up-to-date. Several years ago, Dr. McCormick, speaking in Concord, said that there were then 50,000 doctors in the country who were too poor to practice medicine. That is, they were financially unable to take postgraduate courses, buy books and other helps, attend medical meetings, or even to go on soul- and mind- reviving vacations. Certainly this condition has not grown better in the past five years.

It is evident that the numbers of those newly licensed to practice medicine, over the losses by death each year, is increasing faster than is the growth of the general population. Unless measures are promptly taken by the state to correct this situation, frequent social implications of such a condition would "by comparison render the well-known surfeit of agricultural products relatively innocuous."

Up here in New Hampshire many of our members have had their troubles, but to date our plight has in general been small compared with that of our brethren in the larger cities; we are, however, "on our way", and the socialization of the practice of medicine at times looks pretty attractive to many a doctor hard pressed by the butcher and the baker who have been paid for their wares from the public purse.

For years we have been caring for the sick poor for nothing and have gloried in it. At the same time, however, we have cared for others not so poor and for nothing, and have insidiously developed a group of medical paupers who spend the money they owe us for things we and our families want and often need, and have to go without, simply because we are not good business men.

To this already consuming fire has been added, like highly inflammable oil, the ever-mounting number of free and ten-cent clinics. It has been estimated with considerable accuracy that half a million persons throughout the country are being treated in these free clinics each day. By whom? Why by well-trained conscientious doctors, of course, many of whom walk to their work to save carfare, while the patients arrive at the front door in taxis.

A patient who has acquired the free clinic habit seldom is cured of it, and certainly won't be until a more careful sorting of the really worthy from all others is carried out.

These people are on a dole and have been for years, just as truly as if they were direct recipients in money each day of a part of the million dollars which doctors throughout the country are daily contributing in services for their well-being.

As President Roosevelt says in his latest book, "When any man or woman goes on a dole, something happens to them mentally and the quicker they are taken off the dole the better it is for them during the rest of their lives." Simply because his particular dole happens to be raised by a tax on a particular group, the fun-

damental truth of the President's statement is not altered. Even in Heaven-blessed New Hampshire, there are families, who have never been on town or county aid, yet who have not paid a doctor's bill in three generations.

With the "New Deal" in a "New Era" the sick poor have ceased to be the doctor's problem and have become a responsibility which the whole community must bear.

If nurses, orderlies, and other clinic attendants, to say nothing of supervisors, investigators and relief personnel, are to be paid for their work, doctors must be included when the checks are passed out. Doctors happen to have need of food, clothing and lodging just the same as other public servants, and while much that they learn in the clinics is of value, and the sense of satisfaction in having prolonged life or lessened suffering is beyond recompense, yet it will not buy a new pair of shoes or replace a broken thermometer.

Even now Russia does better for her doctors than is done for American doctors in certain areas of the country.

Only this week I received a letter from one of New York's ablest consultants in which he says, "As to the clinics here in New York; there is no question at all that they are cutting the heart out of the practice of the ordinary physician. Very many doctors, with large practices among those of the lesser economic class, are just about cleaned out."

Again, what are we going to do about it? In his "Imperial Germany" Prince Bulow says, "Despite the abundance of merits and great qualities with which the German nation is endowed, political talent has been denied it."

Substitute medical man for German nation in this phrase and we have an epitome of both the cause of many of our present difficulties and a suggestion for their cure.

We must become more politically-minded, and take a more active part in the formulation of our laws. From a health standpoint we know what the people should have, we appear to be either too lazy or too busy to see that they get it. They cannot afford to let us slip medically, we cannot afford to carry on as we have in the past. We must have a closer knit, better functioning medical body.

It is not unlikely that if medicine could have its dictator as baseball does its Landis, or the movie industry its Hayes, things would go better both for the doctors and the public. We are not likely to get him. Saving this, careful consideration should be given to the advisability of choosing a full-time, adequately paid secretary who could, through his executive ability and personality, control the doctors on the one hand and the public on the other, always letting his left hand know what his right was doing, even if it was the administering of chastisement.

Unintelligent organization will help neither group, and we would better let the deluge come, hoping for a bit of driftwood to catch on to.

We must put the public good first, for if we try primarily to feather our own nests we shall shortly find that we have no nests to feather.

Just as there is the danger in the present emergency set-up of social agencies of permanently pauperizing many of the welfare beneficiaries, so there is the gross risk of a medical slump into stereotyped, non-progressive, mentally pauperizing practice, if the increasing governmental supervision over the health of the community is not guided by actively functioning medical brains.

The things one worries about are not the things which usually happen. Were one omniscient and able to foresee accurately all coming events they could be planned for. If we are involved in the next war there is little question that medicine like industry will be taken over at once by the government, and we doctors in New Hampshire will be regimented along with our fellows of Illinois and California.

In the meantime we have got to rearrange our affairs on a less individualistic basis than the one on which we have been going since medicine began.

According to Rist, this individualism was natural in the infancy of medicine, but "by virtue of its normal development, medicine is about to become social with the force of age."

At times the "New Deal" with its planned economy, its regimentation, its bureaucracy, etc., appears to be getting away from the good old-fashioned poker, and the dealer's choice, not only playing deuces wild, but adding the blond queens and the cock-eyed jacks to "make confusion worse confounded".

In the past few years many people have lost everything that they had of material things. Health alone remains and the doctors have played an important part in preserving that, directly or indirectly.

At times we cannot help feeling like Tommy Atkins, and to paraphrase Kipling,

> For it's old Doc this, and Old Doc that,
> And it's Old Doc you've gone by;
> But it's bless you good kind Doctor
> When the bugs begin to fly.

God knows we can be justly criticized for many shortcomings, but there never was a time when our help was so much needed as at present to plan wisely and deal justly with the vast social and economic problems in connection with our greatest national asset, the public health. This cannot best be done individually or yet as direct employees of the state, but it can be done far better by thorough organization of our profession both with respect to every fellow practitioner and with respect to every individual who comes within our sphere of influence.

Time was when the minister and the doctor guided the thought of the community; a reversion to this status is to be desired, but is not likely of attainment.

Medically to-day's challenge can best be met through a proper functioning of the county societies. The most serious criticism of medicine is not on the professional and technical side, but because of the fact that the administrative and social side has not kept pace with the former. Important changes must be made and made quickly, or large sections of the population will lack adequate medical care and the demand will be met by the state through ill-advised legislation. The State Medical Society, through its component county organizations, can best handle the proposition. There must be no lay interference with professional matters. We want no new social agencies. The people themselves desire the help that we can give them if we will.

They are anxious to know how to keep well, and are quick to take suggestions, but these must be well-considered and meet the economic conditions. First, then, we must have a more smoothly functioning medical organization, and, secondly, we must both individually and as an organization enter more actively and effectively into governmental affairs.

How far do we go toward Moscow, or Rome or Berlin or even toward Washington? Not one step toward any of them in so far as it means a standardized collectivism of medical practice, but all the way if it envisages health, security, and freedom from anxiety for all mankind.

PULMONARY TUBERCULOSIS: MEDICAL ASPECT*

BY ROBERT M. DEMING, M.D.†

I AM pleased to have this opportunity to discuss the medical aspect of Pulmonary Tuberculosis, although, while this is a subject concerning which volumes have been written, necessarily this paper can only briefly outline the more salient features of medical interest.

For centuries Tuberculosis, or "Consumption," in various forms, has been, as its name implies, a consumer of human lives in which process it has conjointly destroyed millions and even billions of potential and actual wealth, and in the treatment of the disease, from both private and public purses, more actual millions have been, and still are, being spent. Hence, Tuberculosis although now slipping somewhat from its age old lofty perch, may still be considered Public Health Enemy Number One in its age group of fifteen to forty-five. What can and may we as the shock troops do to help accelerate its downward trend and, in brief, what has been done?

First, it must be recognized promptly and not as a casual acquaintance, but as a well-known enemy. A brief talk with the patient who seeks our advice, a few cheery words, a bottle of medicine and some pills, accompanied by a pat on the back, or even a casual examination made by listening to three or four places on the patient's chest, frequently over his undershirt, is not sufficient. A diagnosis of "Bronchitis;" "a small spot on the lungs which will soon wear off;" a "run down condition—here's some tonic;" "just a cold—take a few days off;" are words which might be expected from some of the host of medical cubists who, of late years, have arrived amongst us. Nevertheless, our own profession, in haste and with an insufficient examination has, and even now, is subscribing to the above remarks. A friendly talk in which the patient's complaints are brought out, a careful physical examination with the patient stripped, or at least the chest exposed, *repeated* sputum examinations, and if advisable, animal inoculations, and an x-ray by a competent radiologist are essential and valuable to a satisfactory diagnosis. If the complaint is that such an examination takes too much time, I would suggest that an appointment be made for a later date at which more time would be available to you. The patient is highly pleased if you tell him he has no signs of tuberculosis and human nature being what it is, we strive to please. But is it fair, to the patient, yourself, the family or the community? The undertaker has humorously been called "the doctor's best friend." I am tempted sometimes to think the sanatorium is at least a very close friend. From a medical viewpoint, therefore, we are interested in the etiology, diagnosis, treatment and, we must as citizens, be interested also in the economic side of the whole situation.

Tuberculosis is caused by the tubercle bacillus, but this germ in itself, in the majority of cases, fails to produce disease. Unfortunately most of the human race harbor tubercle bacilli somewhere in their bodies, but it takes various other factors to so lower the average human resistance as to allow this bacillus to present symptoms of disease. Such may be lowered vitality resulting from an acute illness such as pneumonia, influenza, grippe, and other respiratory diseases; from unhygienic living and work-

*The first in a series of papers in the Symposium on Pulmonary Tuberculosis read before the Annual Meeting of the New Hampshire Medical Society at Manchester, May 15, 1934.

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ing conditions; modes of living; certain of the trades, particularly the granite and slate workers; overwork, particularly in youth in spite of the adage that "work never hurt anyone;" poverty and its comparative dearth of proper foods, etc.

The method whereby the tubercle bacillus enters the body is still somewhat uncertain, but it may enter through inoculation, inhalation, ingestion, contact, infection through food largely from drinking milk from tuberculous cows (50 per cent of all abdominal tuberculosis in children and 15 per cent to 20 per cent in adults may be traced to this source); carriers and flies.

Climate, I feel, plays but a small part in the causative factor, although some climates favor the development of the disease particularly in the tropics and along the Atlantic Coast; in the one case due to a hot, damp climate, and in the other to the cold, damp winds and fogs. Both sexes are apparently affected about equally, although of late years there has seemed to be a slightly larger proportion of the female sex admitted to sanatoria than previously. This is probably due to extremes in dress characterizing the past decade and to other factors largely connected with social life. Tuberculosis is essentially a disease of young adult life in the age period of twenty to forty. It is not common in children under fifteen years of age, and if present, is apt to occur as the miliary or meningeal form in an acute stage, or with glandular, osseous, or joint disease as a chronic type.

As practicing medical men we are interested largely in the disease from a clinical standpoint. It is not possible for me to discuss tuberculosis of the various organs, most of which conditions are unfortunately diagnosed as incident to some surgical operation or an autopsy. From a radiologist's standpoint it is possible to diagnose tuberculous enteritis, but from a clinician's, a rather lengthy observation period plus the history of tuberculosis elsewhere, is almost a necessity. Tuberculosis of the throat may be readily diagnosed when ulcerations are present, but all too often the early persistent reddening of the anterior pillars is overlooked. I mention these last two conditions largely in an attempt to cover the subject assigned, since they are essentially latter stage complications of the pulmonary lesion. They may of course, be primary but such has not been my experience. Regarding the glandular type of tuberculosis, where the cervical glands are involved, a careful check-up should be made to rule out other causative factors, unless a biopsy is done, such as tonsils and teeth, because so far as I can advise, glandular tuberculosis at first is not dissimilar to enlargement of glands from these sources. A year or so ago I saw an acute case in consultation which, with an uncertain clinical picture and hazy physical findings outside of some moderately enlarged cervical glands, I guardedly diagnosed as acute tuberculous pleurisy. These glands which admittedly I glossed over, on biopsy, evidenced Hodgkin's disease, from which of course, the patient later died. Several cases of tuberculosis of the kidney and one or two of the bladder, have been suggestively diagnosed at the sanatorium, but a definite diagnosis could not be made except by a cystoscopic examination elsewhere. Two cases of tuberculosis of the eye have been observed, but the diagnosis was made elsewhere. There remains the chief of the clan, Pulmonary Tuberculosis.

It has been customary in the past in reaching a diagnosis of early pulmonary tuberculosis to look to the physical examination for a final decision and even now this examination in the majority of cases is all that may be obtained. As a result, percussion and auscultation have been practiced assiduously, and to the skillful they yield essential details. The slightest change in the percussion note and in the character of the breath and voice sounds is readily detected. However, a few rather definite objective symptoms are observed by the examiner or related by the patient. Almost the first symptom a prospective patient complains of is a "tired feeling." This usually follows any exercise which, previously, the individual considered of no import and is accentuated by an increased amount of exercise. No matter how many hours are spent at rest, the "tired feeling" persists. Accompanying this abnormal fatigue the examiner will learn that the disposition, however cheerful previously, is altered and trivial incidents cause a flare-up of temper; that the usual work is not being accomplished so readily and everything frequently "goes wrong." The appetite becomes capricious and requires pampering and a loss of weight is noted. A history of pleurisy with or without effusion may be elicited; there may or may not be fever; tachycardia is a common complaint; and frequently the first symptom the patient notices is the spitting of some blood-streaked sputum or a frank pulmonary hemorrhage. The patient may have consulted you primarily on account of a cough, which he describes as a mild cough, "just like everybody else coughs." Or, he may say he does not cough, neglecting to consider mild cough, clearing the throat in the morning, or hawking as of significance. In many cases the cough in incipient phthisis is annoying at bedtime, disappearing during the first hours of sleep and reappearing during the early morning hours, while after rising it may be severe until the chest is cleared. It may be a good rule to follow, that a person who never coughed, except with a cold, but "caught cold"

for the first time after his eighteenth year, and as a result keeps on coughing for more than a month, is to be seriously suspected of being tuberculous even if there are no definite signs of a pulmonary lesion.

With the cough we must consider the sputum. There is nothing typical about the appearance of the matter expectorated in early phthisis. In the early stage we find that the sputum is scanty, at times, absent. One observer found that in forty-nine per cent of cases in the early stage, the sputum was altogether absent. What is usually coughed up is a viscid mucus, occasionally with some dark specks; it is often frothy and floats on water resembling the expectoration in bronchitis. Whatever the character of the expectoration, do not neglect to have repeated sputum examinations made. I feel it is too often true, that when tubercle bacilli are found in the sputum the case has progressed beyond the incipient stage. A positive sputum makes a possible suspect a positive case, while one or two negative sputa mean nothing. I have dwelt on the subjects of cough and sputum to show: (1) that those who cough are not necessarily tuberculous, (2) that a goodly per cent of those who do not cough are tuberculous and (3) that the character or type of the sputum does not necessarily indicate the severity of the infection.

From our purely physical examination we learn from inspection the shape of the chest, the general build of the individual, the shape of the nails, whether there is any muscular atrophy, whether there is any cyanosis, particularly of the hands, if there is any apparent secondary anemia, or if there are any scars on the neck. Palpation will determine whether on deep inspiration there is any limited motion on one side or the other; the presence or absence of cervical glands and whether there is any increase in tactile fremitus due to possible consolidation. The slightest change in the percussion note should be recognized. It is from auscultation, however, that we derive most of our information. The slightest change in the character of the breath and voice sounds should be noted, although slight apical changes are commonly present even in apparently healthy persons. By slight apical abnormalities I refer to slight changes in the character of the breath and voice sounds and do not include râles. Apical râles have a direct and unquestioned significance in the diagnosis of pulmonary tuberculosis. By râles, I mean the definite moist sounds heard at the apices, or elsewhere, at the end of inspiration and, of chief significance after expiratory cough, not including the various transient "clicks" and other so-called adventitious sounds simulating râles. Definite moist râles at an apex, at the end of inspiration, following expiratory cough, when the other clinical findings point strongly to the presence of pulmonary tuberculosis, practically confirm the diagnosis. Do not neglect the expiratory cough.

Pulmonary tuberculosis does not always begin in the insidious fashion so often pictured. Quite as commonly it settles abruptly on a victim and at once involves such a large area of lung and produces such acute symptoms that the disease has no early stage. There is a widespread impression that pulmonary tuberculosis starts with the eruption of a few tubercles at or near an apex; that these tubercles caseate and allow the formation of fresh tubercles which in turn break down and liberate more bacilli for further implantation; that only indefinite local and systemic symptoms accompany the beginning invasion, the evidence of pulmonary irritation and of general intoxication coming gradually to light with the slow spread of the infection. Fortunately the disease does sometimes develop thus benignly, and when it does, we have an opportunity to use our diagnostic skill to good purpose. However, I am convinced that very often it strikes unexpectedly and without warning, riddling half a lobe, a whole lobe, or a whole lung with tubercles, transporting the patient at a stroke from perfect health to a moderately advanced or far advanced case. This condition may have been caused by the activation of dormant organisms, since statistics show the almost universal infection of man by the tubercle bacillus. Luckily in most of us the infection passes unnoticed, and it acquires clinical importance only when it advances far enough to give disturbing evidence of its presence.

I have made but little mention of the x-ray in the diagnosis of pulmonary tuberculosis for several reasons. First, because of the expense involved. Many patients are unable to finance this procedure, even when available. Secondly, while the roentgen ray portrays slight pulmonary changes with remarkable precision and gives an objective and permanent record of conditions, the same difficulties surround it as stand in the way of a diagnosis through finesse in percussion and auscultation. A large proportion of healthy adults show apical abnormalities on physical examination, and an equal or larger number show abnormalities in density to roentgen ray illumination. The anatomical interpretation of plates is not difficult, but the clinical interpretation is difficult, because they give no etiological or physiological information and, therefore, to a large extent fail to distinguish between an active and an inactive lesion. The x-ray tells us only of density, whereas auscultation discloses moisture, and in this respect the x-ray has all the shortcomings of a physical examination. It, nevertheless, is of the greatest assistance as a comparative check with the physical

and other examinations. If the x-ray shows lesions at the apex, the case is probably fairly well advanced (Kirklin). The following five points given by Dr. Lawrason Brown will prove of much value in arriving at a diagnosis; with the exception of the first which is self-sufficient, at least two of the others must be present:

1. The presence of tubercle bacilli in the sputum.
2. Persistent râles at one apex.
3. X-ray showing involvement over same area.
4. History of hemorrhage of at least one teaspoonful of clear blood.
5. Pleurisy with effusion.

The subcutaneous use of tuberculin as a diagnostic agent has its many advocates and opponents. In brief, I can only say that a positive tuberculin reaction indicates the fact that at some time, somewhere, the patient has been infected. It does not prove the activity of the infection. The value of the positive test in infants and very young children, as a diagnostic aid, is considerable and that of the negative test in suspects of all ages can hardly be overestimated since it practically excludes tuberculosis past and present. Known tuberculosis cases, which do not react, are almost always obviously far advanced terminal cases.

Having briefly and rather sketchily treated the subjects of etiology and diagnosis our next consideration must be that of treatment. Only various modifications of physical rest, combined with mental rest, good, nourishing food, fresh air and sun have entered into this picture since the original formula was promulgated years ago. True, in the early days, the "roughing it" as then advised, seems quite harsh as compared to our present scheme and largely resulted in the survival of the fittest, but, at least, it did attain satisfactory results and was a big step forward over the previous hit or miss medicinal treatments in the course of which practically all the drugs in and out of the pharmacopoeia have been utilized. To my knowledge, there is no medicine or formula which exercises more than a more or less passive influence on the course of tuberculosis, and many of them in time, do some damage to the all-important digestive apparatus. I except our old friend cod liver oil and its recent substitutes, although we should, strictly speaking, consider this substance as only a food. Its use, however, with tomato or orange juice, as suggested by McConikey of Saranac, in the treatment of intestinal tuberculosis, has been used quite extensively by us in connection with heliotherapy, with most gratifying results. We have not used tuberculin as a treatment, although by some men satisfactory results have been reported, chiefly in glandular cases. The gold treatment using such preparations as sanocrysin, has not achieved much popularity largely due to the danger involved in its use, and its only very moderately successful results when a series of cases were treated. In Germany the salt-free diet has offered encouragement, but the chief difficulty has been to control the dietary desires of the patient. My impression is that efforts to produce an intravenous medication to an end to dissolve the protecting waxy capsule surrounding the bacillus and kill the germ, have been largely dropped due to the fact that it is now recognized that tuberculosis is not a blood-borne disease and hence treatment through the blood stream would be of no avail. Various vaccines have offered some encouragement. More efforts are now being made to effect some attenuation in the growth of the tubercle bacillus. Heliotherapy, using the direct sun rays or various lamps, is useful in some tuberculous conditions, but should be used almost anywhere else in pulmonary lesions than on the chest. Balanced meals consisting of good, nourishing, well-cooked, plain foods, are most essential in the treatment of almost all bodily ills and chiefly so in tuberculosis. Pulmonary tuberculosis being essentially a toxic condition, burns up the bodily tissues and easily assimilated fuel is necessary to maintain bodily resistance. Pampering of the patient's appetite is a grave mistake, as is urging the patient to eat between meals except in the case of a very poorly nourished individual. With in-between meals, the digestive apparatus has practically no opportunity to rest and a good many years' experience in tuberculosis work has cautioned me to treat the alimentary tract with great respect. Fresh air, not necessarily cold air, is a rather obvious requirement.

For patients suffering from tuberculosis, in practically any form, bed rest is essential in graduated doses. Those running a temperature or having persistent, productive or non-productive cough, increased pulse or respiration on light exercise, loss of, or no gain in weight, should be in bed all of, or the greater part of, the time. Such routine, regulated rest, and I wish to emphasize this system of rest, and its accompanying dietary regulations, may be well carried out at most homes, but it seldom is. I am connected with a sanatorium, but if I were not, with my present information, I would strongly urge any patient with tuberculosis to enter one. "Misery loves company" is no better illustrated than is the reaction of the average patient following the first few days after entering the sanatorium. Group treatment as must be carried out in a general way in most sanatoria, has its disadvantages, but I feel that its advantages over home treatment, in most cases, are great. Regular rest may be maintained, well-cooked nourishing food in sufficient variety may be obtained without pampering an already capricious appetite, special-

ized treatment is available, the patient is not made to feel that he is a pariah among his associates, proper nursing is afforded, contact danger to others of the family avoided, and of considerable importance to the patient and his family is the fact that sanatorium treatment may be obtained for him at nominal or no expense.

Since rest is the essential part of treatment, it stands to reason that any method or methods which will attain rest, to the ultimate degree, are justified. Bed rest, rest obtained through braces or casts, rest for a tuberculous larynx through silence, or rest to the lungs by use of artificial pneumothorax, oleothorax, phrenic nerve crushing or exeresis, pneumolysis, or extrapleural thoracoplasty are methods used. Bed rest combined with as much freedom from mental worry as is possible, is, and has been, standard sanatorium treatment, and practically all cases should receive this primarily. In addition to such rest, acute progressive cases, those with cavitation, and those patients who do not show improvement from bed rest alone, should receive the benefits of other of the methods previously mentioned. Since this paper proposes to cover the medical aspect only, the pure surgical procedures I will leave to my surgical associate, Dr. Overholt, whose assistance, together with other members of the Lahey Clinic, has allowed the State Sanatorium to offer its patients all the benefits of modern treatment.

Artificial pneumothorax as elaborated by Forlanini about 1912 and as utilized in this country for, I believe, the first time about 1914 by Cleaveland Floyd of Boston is increasing in popularity after a rather slow, hesitating start and is now widely used and its value recognized. I might note that at Glencliff some 3,000 initial treatments and refills have been given during the past two years and treatments are averaging 125 per month. The indications for pneumothorax may be divided into those which show necessity and those which show expediency. It is necessary when bleeding from the lungs is repeated and voluminous, also when tuberculosis in one lung is rapidly progressive. It is expedient in chronic progressive cases, or cases in which there are periodic lapses into activity with the disease localized to one side, and when as observed previously, other forms of treatment appear to exercise no restraining influence. It is also expedient in chronic, progressive cases or chronic periodically progressive cases with cavity formation, much fever and toxemia even when there is evidence of activity in the other lung, of a limited amount. It is expedient to carry out the operation to replace a serous pleural effusion occurring in young adults, even though there may be no evidence at the time of the effusion or disease in the lung on the same, or even the opposite, side. If there are bilateral signs of disease in the lungs, we can only be guided to the lung which should be collapsed, by the evidence, clinical or radiographic, of the existence of a cavity; if no cavity can be found then a trial should first be made of collapsing the lung where the greater amount of pathology is present. Artificial pneumothorax should not in any sense be considered a cure, it simply is a valuable adjunct to the routine bed-rest treatment. Its primary object, as is true of any collapse therapy, is to close the cavities and eliminate tubercle bacilli from the sputum. The technique of the operation is comparatively simple, the patient need not be hospitalized, although this is advisable during the early part of the treatment, the apparatus is very moderate in price, no special training is necessary, and such treatment may be given by any careful physician in his office or at the patient's home. X-ray plates should be taken or fluoroscopic examinations made, at frequent enough intervals so that the physician may keep informed as to the extent of collapse obtained and whether any fluid is forming; such complication occurs in approximately thirty per cent of all cases.

To sum up, tuberculosis from a medical aspect, must be considered from a diagnostic and a treatment viewpoint. It must be diagnosed from observation, history, physical, roentgenological and bacteriological examinations, and treated by routine, regular mental and physical rest, obtained at the home or sanatorium and assisted by one or more of the various methods previously mentioned and explained briefly in this paper and to be explained by the following speakers.

EDITORIAL NOTE: Other articles included in this Symposium will be published in later issues. The discussion (which applies to the entire Symposium) will appear following the last article of the series.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.

CASE 20371

PRESENTATION OF CASE

A sixty-six year old Italian stone carver entered complaining of epigastric pain of three weeks' duration.

Eleven years before admission after an x-ray diagnosis the patient received dietary treatment for ulcers of the stomach. The treatment lasted six months following which he had no complaints. Two years before admission he began to have occasional mild epigastric pains. He paid very little attention to them. Four months before entry his family noticed that he did not eat so much as usual. One month before entry he developed a feeling of fullness in the epigastrium, immediately after eating, which lasted for several hours. For the past two weeks the epigastric pain came on immediately or about an hour after meals and radiated to both upper quadrants. At the same time there was a dull ache in the lumbar region. The pain lasted for about an hour and then stopped. It was often relieved by lying down with a pillow under his back.

The family and past histories are non-contributory.

His wife and three children were living and well.

Physical examination showed an emaciated man lying in bed in no acute distress. There was moderate cyanosis of the lips and fingertips. The teeth were dirty. There was marked pyorrhea. The tongue was dry and beefy red. The tonsils and pharynx were dull red. The chest was slightly barrel-shaped and showed a moderate number of moist râles, especially at the right base. The heart was not enlarged. The sounds were of fair quality. There was a soft systolic murmur over the precordium. The blood pressure was 100/76.

The temperature was 99°, the pulse 84, the respiration 20.

Laboratory examination of the urine showed a slight trace of albumin and a specific gravity of 1.018. Examination of the blood showed a red cell count of 8,010,000 and a hemoglobin of 95 per cent. The white cell count was 10,000, with 87 per cent polymorphonuclears. Six subsequent red cell counts ranged from 6,500,000 to 7,500,000. The first stool consisted of about 200 cubic centimeters of dark red blood with only a small amount of fecal material. All the following stools showed positive guaiacs.

On the day following admission he vomited about an ounce of blood. During the next few days he had no further bleeding. On the fourth day the right parotid began to swell. This swelling increased and became tender. The temperature rose to 101°. On the fifteenth day incision and drainage of a parotid abscess was performed. A culture showed staphylococcus aureus. About one week following operation he began to go downhill, his temperature rose to 103.5° and he died approximately one month after admission.

DIFFERENTIAL DIAGNOSIS

DR. F. DENNETTE ADAMS: The occupation introduces the probability of silicosis with possible tuberculosis. The history of recurrence of symptoms after a period of freedom is quite characteristic of ulcer. Usually the interval is shorter than nine years but occasionally it is even longer. It is not unlikely that once symptom free after the first attack, he gave up his dietary treatment as many do under the mistaken impression that the disease was cured. In these cases the recurrences may be due to dietary indiscretions alone or there may be contributing factors such as intercurrent infection, alcohol, or very commonly added physical or emotional drain. On the other hand there is not infrequently recrudescence of symptoms without demonstrable cause, even when medical treatment has been faithfully continued.

The failure to eat as much as usual is more characteristic of cancer than of ulcer in the absence of obstruction, provided failure to eat is due to actual loss of appetite and not to fear of eating. On this point the record is not clear.

The feeling of fullness in the epigastrium after meals is more characteristic of cancer, but could be due to pyloric obstruction. Radiation of the pain to both upper quadrants does not give any added leads.

If the dull ache in the back had been in the dorsal region it would introduce the possibility of penetration of a duodenal ulcer. Appearing in the lumbar region it could best be explained, if one expects to account for it on the basis of gastrointestinal pathology, as due to metastatic cancer; but it could well be caused by some unrelated condition such as back strain or chronic arthritis. One of the latter would better account for its periodicity and the fact that lying down with a pillow under the back provided relief.

The cyanosis and the râles at the base were probably associated with silicosis.

The systolic murmur over the precordium is a common finding at sixty-six, being due to

roughening of the arch, relative mitral insufficiency of an aging myocardium, or both.

There is no record of abdominal findings; so one must assume that nothing of importance was found. A small palpable mass, if present, would add very little as it could be due either to obstructive scar tissue at the pylorus or to cancer. A mass high up or on the left would be more suggestive of the latter. One must assume also that rectal examination was negative and that there were no demonstrable signs in the back; some tenderness or limitation of motion would be expected.

A red cell count of 6,500,000 to 8,000,000 is most unusual in a man who is bleeding. Dehydration could hardly account for it. Poor pulmonary ventilation based on silicosis with or without emphysema would seem the most likely explanation, unless there is a true polycythemia present. The only added clue which might point toward the latter, a large spleen, presumably was not found.

Inflammation of one or more of the salivary glands is common as a terminal event in cachectic patients.

The patient gave a history of previous ulcer of the stomach. From the standpoint of differential diagnosis it would be extremely valuable to know whether the lesion was actually in the stomach, fundus, or prepyloric region, or in the duodenum. A report of the x-ray taken eleven years before admission would supply this valuable information. If this lesion were limited to the duodenum we would be justified in discounting the possibility of malignant change; whereas the opposite would be the case if the original lesion had been in the stomach, and we would think in terms of cancer. There was probably no obstruction since the vomitus appeared to be pure blood. In an obstructed case one would expect evidence of stasis in the vomitus. Gastric analysis was not performed, doubtless because of fear of increasing hemorrhage; so we lack the evidence which this procedure would have provided.

The bleeding and loss of appetite could be present with either ulcer or cancer. The back pain, not the type seen in penetrating ulcer, might be due to metastatic cancer but its character is more suggestive of some independent condition.

It is impossible on the evidence available to make a flat-footed diagnosis. One could say ulcer and not be condemned if autopsy showed cancer, and the converse is equally true. My guess would be posterior duodenal ulcer, although between this condition and cancer it is a pure guess. Terminal septicemia, abscess of the parotid gland, silicosis and arteriosclerosis undoubtedly were present. Terminal bronchopneumonia is always likely.

Clinical Discussion

A Physician: Might the high red cell count have been due to dehydration?

Dr. Adams: It is unusually high for that.

A Physician: He had a concentration in the urine of only 1.018. If there is a dehydration erythremia would not the specific gravity of the urine be higher?

Dr. Adams: Yes, I think it would.

A Physician: Do you believe carcinoma frequently develops upon ulcer of the stomach?

Dr. Tracy B. Mallory: The opinion is varied on that. We do not believe very many ulcers become malignant, but sometimes there is not the slightest doubt. Dr. Hampton in the x-ray department has made a very interesting observation in the past few years that if there is a lesion in what he calls the prepyloric region, the last two centimeters before the pyloric ring, that lesion will turn out to be cancer almost every time even if it has all the x-ray findings of ulcer. We have had all told about twenty-five cases of "prepyloric ulcer" of which only two were benign. Three of them were the earliest cancers of the stomach I have ever seen, really precancerous lesions; so that I think in that particular region these lesions start as neoplasms and secondarily ulcerate.

Dr. Adams: We are no longer willing to treat for a prolonged period any case of ulcer situated in the stomach which does not show fairly rapid and definite improvement symptomatically and by x-ray, because of fear that it will degenerate.

Dr. Mallory: Dr. Holmes feels, and I agree with him, that they are too reluctant to examine these patients with x-ray. It would of course be dangerous to give them barium meal and poke them very hard; but if they knew it was a bleeding case they could examine it with unusual care. It is extremely important to know whether the lesion is in the stomach or duodenum. Both prognosis and treatment are quite different, depending on the location. It is surprising, too, that no more interest was aroused by his high red cell count. He was transferred to my department with a clinical diagnosis from the hospital of a bleeding ulcer, agreeing with Dr. Adams, and of a parotid abscess and terminal bronchopneumonia.

Clinical Diagnoses

Bleeding ulcer.
Parotid abscess.
Terminal bronchopneumonia.

Dr. Adams' Diagnoses

Bleeding duodenal ulcer.
Parotid abscess.
Silicosis.
Bronchopneumonia, terminal?
Septicemia, terminal?
Arteriosclerosis.

Anatomic Diagnoses

Carcinoma of the stomach.
Parotitis, suppurative, right.
Degeneration (? old infarct) of left occipital lobe.
Bronchopneumonia.
Pleuritis, chronic fibrous, bilateral apical and left diaphragmatic.
Arteriosclerosis, marked coronary, aortic and celiac.
Hydrothorax, bilateral.
Perisplenitis, marked.
Intussusception, terminal.
Infarction of adrenal, early.
Pedunculated polyp of the sigmoid.
Cystitis.
Operative wound, incision and drainage of parotid abscess.
Polycythemia vera.

Pathologic Discussion

Dr. Mallory: What we actually found was a carcinoma of the stomach on the lesser curvature 12 centimeters up from the pylorus and well up from the pyloric zone. It was quite large, eight centimeters in diameter, with a central area of ulceration from which hemorrhage had occurred.

Dr. Adams: There was no silicosis?

Dr. Mallory: No; though I think that was an ingenious and reasonable guess. The lungs were negative except for pneumonia. The heart was quite small, weighing 210 grams, and the right ventricle measured only three millimeters which is less rather than more than normal. The spleen on the other hand weighed 500 grams and I think should have been felt clinically, because with hemorrhage it probably had decreased considerably in size. The bone marrow showed a very marked red cell hyperplasia. So I must agree with the man who suggested polycythemia, though we are probably dealing with the disease at an early stage.

A Physician: He had emphysema, did he not?

Dr. Mallory: No.

A Physician: The chest was slightly barrel-shaped and showed a number of moist râles at the right base. Barrel-shaped chests usually mean emphysema.

Dr. Mallory: Most chests at sixty-six are barrel-shaped or begin to be and they may or may not have emphysema. One has to have more than that to make a diagnosis of emphysema. I think there is very little doubt that there was polycythemia but that was only incidental in this case. The real cause of his death was carcinoma of the stomach. It is interesting in this connection that polycythemia has been found very frequently in association with ulcer of the stomach, though not so far as I know with cancer. Boyd* has recently published a case report and a bibliography bearing on this.

*Boyd, W. Am. J. Med. Sc. 187:589, 1934.

CASE 20372

Presentation of Case

Approximately three years before entry the patient, a thirty-two year old single American farmer, developed frequent nausea and vomiting after breakfast. There were no other symptoms. Six months later he began to have frequent loose bowel movements containing blood and mucus. These bowel movements increased in frequency, averaging ten to twelve a day. He was a little weak but kept on working. His condition improved in a few months and for the next eight months he had only one to three bowel movements a day. The stools were formed and contained only occasional small amounts of blood and mucus. About a year and a half before entry his diarrhea returned. He had ten to fifteen stools a day and two to four at night. The stools contained blood and mucus. He had anorexia, weakness and loss of weight. He entered a hospital where he was put on a diet and given irrigations. He remained there for two weeks and after discharge was in bed at home for eighteen weeks. During this period he had from ten to thirty stools a day. His weight dropped from 240 to 120 pounds. One year before entry he was able to do a little light work. He slowly gained in weight and strength and had only two soft stools a day with only occasional blood and mucus. During this period he remained on a strict diet, took a tablespoonful of mineral oil each morning and a saline enema each night. He gained in weight and did fairly well until five months before entry when the diarrhea increased. It was not very severe however until two months before entry, when he had a large bloody movement. Since then he rapidly went downhill. He had ten to thirty movements a day associated with cramps and tenesmus. He remained in bed for seven weeks and lost about thirty pounds in weight. Ten days before entry he had many more large bloody movements with the loss of a pint to a quart of blood. He felt weak but did not faint.

The family history was negative; no history of tuberculosis.

The past history is non-contributory.

Physical examination showed a well-developed, poorly nourished pale man lying in bed in no acute distress. The skin and mucous membranes were pale. A few teeth were absent. The remainder were dirty and showed some caries and pyorrhea. His heart was normal in size. The sounds were rather muffled. There was generalized moderate tenderness over the whole

abdomen, especially over the course of the colon in the left lower quadrant. The tip of the spleen was just palpable on inspiration. Upon rectal examination the mucous membranes felt thickened and thrown into folds. The blood pressure was 124/60.

The temperature was 99.8°, the pulse 112. The respirations were 20.

Laboratory examination of the urine was negative. Examination of the blood showed a red cell count of 2,900,000, with a hemoglobin of 45 per cent, and a white cell count of 22,000, with 88 per cent polymorphonuclears. A smear showed slight variation in the size of the red cells and moderate achromia. The platelets were normal in number. Many stool examinations were made all of which showed four plus guaiacs. The stools were soft, very often watery, and contained at times some mucus and pus. No amebae were found. A Hinton test was negative. The non-protein nitrogen of the blood was 31 milligrams, the serum protein 5.2 milligrams. A tuberculin test of 1-1000 was negative. Agglutinations for typhoid and dysentery were negative.

A barium enema showed a small rectum. There was absence of haustral markings and irregular contours to the entire bowel with a definite defect in the cecum measuring 2 centimeters in diameter. There were numerous ulcerative deformities of the rectosigmoid and splenic regions. There was also a pressure defect at the splenic flexure. A chest film was unsatisfactory in that the apices were not well shown. The lower portions of the lung fields were clear.

He was put on a first stage colitis diet and given bismuth subcarbonate every four hours. On the fourth day he had a large hemorrhage of about 400 cubic centimeters and another on the sixth day. He was transfused following each of these hemorrhages. On the eighth day an ileostomy was performed. He developed some edema of the ankles. On the eighth postoperative day his temperature, pulse and respirations began to rise. He became disoriented. There was some bronchial breathing and dullness on the right and scattered râles over both bases. He died early that evening.

Differential Diagnosis

Dr. F. Dennette Adams: The history of severe diarrhea with blood and mucus of three years' duration points to disease of the colon. The remissions are characteristic of ulcerative colitis and usually are not seen in tuberculous disease, which is more likely to show definite improvement under treatment or a more steadily downward course. Amebic dysentery is possible but rarely produces in chronic cases such marked loss of weight and incapacitating weakness. Patients with this disease are never entirely well but are able to get about, to work, and to lead fairly normal lives. Their movements are not so frequent. This is not necessarily true of the acute cases, but here we obviously are dealing with a chronic disease.

In considering any lower bowel disorder, malignant disease must be excluded. Most of us are taught that cancer is a disease of middle or advanced age, but this does not pertain to cancer of the bowel. We are constantly seeing it in the third or fourth decades, in fact it is a common condition during these years.

Dr. Tracy B. Mallory: There is a case on the ward now of typical cancer of the rectum in an eleven-year-old girl.

Dr. Adams: The duration here is long for cancer without at least some evidence of obstruction. Remissions are not apt to occur. I do not believe cancer is present in this case, but it would be necessary to consider it even more seriously if there were no remissions, if constipation were alternating with diarrhea, and if abdominal pain or cramps were in the picture. On the other hand one must not be misled by absence of constipation and exclude cancer for this reason. Not infrequently constipation occurs only in the late stages, for the disease may be well advanced before partial obstruction begins. I have recently seen a man who had had diarrhea for only three weeks followed by constipation for one week. X-ray examination revealed a questionable lesion in the ascending colon and he was explored for possible early cancer. Operation revealed extensive inoperable cancer in this region with metastases in the liver. Death occurred within a month, from cancer.

This discussion of cancer is introduced here in order to emphasize the importance of its consideration in any patient with symptoms referable to the colon and particularly to call your attention to its frequency before the age of forty, as well as after.

In a differential diagnosis of chronic diarrhea, diverticulitis must be excluded. Against it are the presence of blood, absence of pain or cramps, and the frequency of movements. Localized tenderness with or without a palpable mass is also usually present, and one expects some evidence suggestive of an acute or subacute surgical condition. Diverticulosis without an acute episode would not give such severe symptoms or so much systemic reaction as this case presents.

Chronic diarrhea always suggests deficiency disease of some type; but failure to respond to what was presumably good treatment elsewhere, the presence of so much blood, and the absence of other signs, exclude such a possibility.

In this case on the basis of the history alone ulcerative colitis, tuberculous colitis and amebic dysentery, in the order named, would be the most likely possibilities.

"Physical examination showed a well-devel-

oped, poorly nourished pale man lying in bed in no acute distress." I am surprised that no mention is made of evidence of marked loss in weight, which must have been present in a man who has dropped from 240 to 120 pounds.

"The heart sounds were rather muffled." The sounds may have been poorly heard because he was so thin that when the examining physician put a stethoscope down on the chest wall it was simply resting on top of a rib.

"Examination of the blood showed a red cell count of 2,900,000." Secondary anemia probably, and readily understandable.

"No amebae were found in the stools." This by no means excludes amebic dysentery; but it is a point against it.

"Agglutinations for typhoid and dysentery were negative." Will Dr. Mallory tell us the value of agglutination reactions in bacillary dysentery cases?

DR. MALLORY: In bacillary dysentery a positive result is valuable.

DR. ADAMS: "Barium enema showed a definite defect in the cecum measuring two centimeters in diameter." This is the region in which we expect to find tuberculosis.

"There were numerous ulcerative deformities of the rectosigmoid and splenic regions." This is characteristic of ulcerative colitis.

"A chest film was unsatisfactory." This should have been repeated to exclude the apical lesions of tuberculosis.

Proctoscopic examination is not mentioned. It probably was done.

The differential diagnosis lies between ulcerative colitis and tuberculous colitis with the evidence in favor of the former. Terminal bronchopneumonia was the immediate cause of death.

A PHYSICIAN: Would the fact that his weight dropped from 240 to 120 pounds have any bearing on tuberculosis?

DR. ADAMS: I think not. Ulcerative colitis can proceed slowly downhill or go very rapidly and loss of weight is not inconsistent with either condition. Amebic dysentery has been ruled out as well as one can rule it out. In a case of this kind, in view of the recent increase in cases of amebic dysentery, one would be justified in the absence of definite diagnosis in giving a course of emetin as a therapeutic test.

The most important differential factors in this case are the progress by remissions and the x-ray evidence of most extensive damage in the distal rather than the proximal colon.

DR. MALLORY: What would you suggest in the way of treatment for him?

DR. ADAMS: Bed rest; a low residue, high vitamin, high caloric diet, with frequent feedings; one or more test courses of emetin; and as was done in this case, surgery if medical treatment failed to produce results. This man probably did not get emetin, as we were not so much on the alert for amebic dysentery when he was in the hospital a year ago.

A PHYSICIAN: Do you find that these terminal bronchopneumonias after diseases of the colon are due to emboli similar to those that you find after diseases in the prostate, with thrombosis of the prostatic plexus.

DR. MALLORY: No, I should say not. They are usually a combination of collapse and bronchopneumonia.

A PHYSICIAN: What medication do you use to control spasm and diarrhea?

DR. ADAMS: The favorites are belladonna, paregoric, tincture of opium, but many others are tried from time to time.

CLINICAL DIAGNOSES

Ulcerative colitis.
Bronchopneumonia.

DR. ADAMS' DIAGNOSES

Ulcerative colitis.
Bronchopneumonia.

ANATOMIC DIAGNOSES

Ulcerative colitis.
Operative wound, ileostomy.
Generalized peritonitis.
Perforation of the ileum?
Bronchopneumonia.
Arteriosclerosis, slight aortic.

PATHOLOGIC DISCUSSION

DR. MALLORY: This man showed a perfectly typical ulcerative colitis. The entire large intestine starting just above the anus and running all the way around the cecum and actually up the ileum for four or five inches showed innumerable small ulcerations into the muscularis with intervening shaggy projections of the mucous membrane. The lesions of ulcerative colitis characteristically are most marked in the rectosigmoid and get less and less marked as you get around to the cecum. Sometimes we find lesions only in the descending portions of the colon, none in the transverse or ascending parts. In the later stages or quite early in the more fulminating, rapidly progressive ones the process may extend all the way around to the cecum and occasionally involve the ileum. The whole disease can run its course in an astonishingly short period. Death can occur within less than three weeks after the onset, or it may be an extraordinarily chronic thing and last for years. It is quite characteristic that it should have the remissions that this case showed.

One point in the pathologic changes deserves emphasis. In certain stages the mucous membrane between the ulcers may be so thickened with inflammatory infiltration and hyperplastic proliferation that it may become difficult to make a diagnosis between ulcerative colitis and

multiple polyposis on a proctoscopic examination. I brought this specimen to show you what polyposis may be like. This of course is a rare disease, at least in the extreme grade that involves the colon from end to end with hundreds of polyps. The differentiation of the two diseases would depend mostly on the proctoscopic examination. With polyps as big as some of these perhaps they could make a diagnosis, but more often I have seen cases in which the diagnosis was confused on proctoscopic examination. In the multiple polyposis cases there is, however, no true ulceration between the polyps. The mucosa is normal between the polyps and enormously hypertrophied in the polyps.

An important reason for separating the two conditions is that the prognosis is entirely different. Ulcerative colitis is an inflammatory disease. I rather think it is infectious but I do not know that it is. Polyposis is a neoplastic disease with the constant danger illustrated in fact by the case I am demonstrating, that malignancy will supervene and metastasis occur. Obviously ileostomy in such a case is a futile gesture, and complete colectomy is the only type of therapy that is worth trying. In ulcerative colitis an ileostomy will often give a symptomatic cure but sometimes it fails and we are beginning to resort to a complete colectomy in ulcerative colitis as well.

DR. ADAMS: It is astounding how acute some of these cases can be at onset. Severe diarrhea, with profuse bleeding, high fever with marked prostration and systemic reaction, not infrequently usher in the disease. Proctoscopy of such patients is of no value, as extensive hemorrhage prevents adequate examination. Ileostomy is usually the only hope in these fulminating cases and this may be of no avail. If the patient recovers from the acute stage, extensive damage to the colon usually remains and we have as an end result the typical picture of chronic ulcerative colitis.

DR. MALLORY: An important thing to remember is the likelihood of perforation of one or more of these ulcers. It is a very common cause of death. Nowadays it is a much more common type of intestinal perforation than typhoid.

The New England
Journal of Medicine

SUCCESSOR TO
THE BOSTON MEDICAL AND SURGICAL JOURNAL

Established in 1828

Published by THE MASSACHUSETTS MEDICAL SOCIETY under the jurisdiction of the

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SUBSCRIPTION TERMS: *$6.00 per year in advance, postage paid for the United States, Canada $7.04 per year, $8.52 per year for all foreign countries belonging to the Postal Union.*

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 8 Fenway.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to The New England Journal of Medicine, 8 Fenway, Boston, Mass.

FADS, FASHIONS, AND FALLACIES IN MEDICINE

IT is a familiar and recognized tendency of the human mind, especially the American mind, to seize upon any new thing and make a fad or fashion of it, merely because it is new, irrespective of whether, upon sober consideration, it promises to prove rational or fallacious. This characteristic may be observed not only in politics and in the sartorial arts, but in hygiene, in medicine, and in the other sciences as well. In the field of medicine it is exhibited chiefly by the laity, yet not by them exclusively. Sometimes, some doctors, in spite of their professional training, are not immune from the disposition to make a fad of some item of technique or treatment, or some bit of laboratory suggestion.

Examples of fashion in medicine may be found in the fields of dietetics, of obstetrics, of pediatrics, and of surgery. One of the prevalent dogmata in the feeding of children, is that every child must drink a quart of milk and eight ounces of orange juice daily in order that his teeth may grow hard and strong. Whatever the laboratory evidence on which this theory is based, it is probably true that heredity plays a larger part than diet in the characteristics of the teeth, as of the other tissues of the body. In a family of children all fed alike, where one parent has good teeth and the other poor, the qualities of the children's teeth will follow Mendel's law, irrespective of diet. Now milk is not a natural beverage of the human being, or of any other mammal, after the period of infancy. It may be that the human race has become, in a sense, parasitic on the cow; but this parasitism, due to inability or unwillingness of the human female to fulfill her function, need not extend beyond the first year of life. Some children, after drinking the milk and orange juice which are their required quota for breakfast, have little appetite or room left for the bacon, bread, cereal, egg and fruit which should be the chief staples of their meal.

When vitamins were first discovered and their relation to deficiency diseases noted, the idea was promptly seized by the laity and commercially exploited by the trade for profit. In the popular mind the importance of vitamins has been grossly exaggerated, and their use carried to excess. Because an adequate amount of the various vitamins is essential to good health is no reason why every human being should be consistently overdosed with them from birth. A little whiskey may be good for one occasionally, but it is not a desirable or exclusive essential of every meal. Other fruits contain vitamin besides oranges.

The same commercial phenomenon may be observed in the matter of cereals. It is questionable whether any cereal, except possibly rice, is superior or equal as a food to oatmeal,—one of two things which have made the Scots a hardy race. The multitude of breakfast foods which most people eat in lieu of oatmeal are for the most part inferior and expensive substitutes foisted upon the gullible public by advertising methods which create a fashion or fad in their favor.

In obstetrics and in surgery a variety of fashions within the past forty years have had their little day and ceased to be. Scopolamine-morphine anesthesia, the fad of a generation ago, is now in the way of being discredited, after costing the lives of a good many babies. The barbiturates are the fashion of to-day, and may be no more permanent. Even the use of ether has been overdone in obstetrics. Universal episiotomy or universal version are no more likely to prove desirable than universal tonsillectomy or universal circumcision. Thirty years ago nephropexy was the fashionable operation upon women. Few things in this world are momentous enough to be universal except death and taxes.

In all these phenomena we see merely mani-

festations of the common mental process of unconsidered generalization, the tendency, from which even some physicians are not exempt, to seize upon any new drug or operation or technique or theory, and apply it to all sorts of conditions to which it may not be adapted. Thus arise the fads, fashions, and fallacies of medicine. It is the old dogma of the panacea, the tendency to believe that any new thing which may be good or true under certain circumstances can be universally applied with benefit. Of course new ideas must be tested, but with a wholesome skepticism, a resolution to hold fast only that which proves to be correct, and its corollary to abandon that which has been serviceable only when it is clearly demonstrated that something else is better. By such an attitude the doctor may avoid the temptation to be "blown about by every wind of fashionable doctrine", bearing in mind rather Pope's admirable and Polonius-like maxim:

"Be not the first by whom the new is tried
Nor yet the last to lay the old aside."

EPIDEMIC MYALGIA

WITH the improvement in diagnosis and epidemiological control, new diseases, or at least newly recognized diseases, arise from time to time. One of the most interesting of these to be brought to our attention recently is "epidemic myalgia" named by Sylvest[1], who presents a very thorough study of the condition. His attention was first drawn to the disease during an epidemic on the island of Bornholm in the Baltic Sea occurring in 1930. Since then some 10,000 cases have been noted in Denmark.

The condition is characterized by sudden onset, its occurrence in the summer, its tendency to relapse, and intense muscular pain associated with tenderness and some swelling of the involved muscles. Those of the back, abdomen and the diaphragm seem to be particularly frequently involved. Indeed the pain may be so severe as to resemble renal colic.

A few cases answering this description have been noted in Boston recently. Sylvest in a very thorough study of the subject has noted the similarity of his cases to the scattered reports of epidemics resembling dengue fever and the so-called "epidemic pleurodynia". One of these American epidemics was reported from Cape Cod by Churchill, Landis and Glusker[2]. They observed fifty cases, with the outbreak beginning in July, 1925 and ending in early September.

The etiology is uncertain, but the disease is apparently spread by contact infection, and the occurrence of herpes in some of the cases suggests an association with a virus disease.

In addition to this country, where the first cases were apparently described by Dabney in 1888, and in Denmark, it has been noted in Iceland, England, Germany, Sweden, Finland and Portugal.

The suggestion of Small[3] that the disease might be due to a plasmodium has not been confirmed. The few cases that have been noted here emphasize the need of considering this condition in the differentiation of acute abdominal, pleural or low back pain. Ultimately we may hope to learn more about the character of the disease and its behavior. On the basis of the Danish experience it must be considered as readily transmitted by contact and possibly belonging to the rather too little known group of the virus diseases.

REFERENCES

1 Sylvest, Ejnar: Epidemic Myalgia. Bornholm Disease. Copenhagen, 1934.
2 Churchill, F. S.; Landis, E. M., and Glusker, S. D.: An epidemic of undetermined nature: Dengue? J. A. M. A. 87: 821, 1926.
3 Small, J. C.: A protozoan organism within the erythrocytes of patients suffering from epidemic pleurodynia. ("Devil's grip.") Am. J. M. Sc. 168: 570, 1924.

THE TENTH CLINICAL CONGRESS OF THE CONNECTICUT STATE MEDICAL SOCIETY

THE announcement of this Congress appeared on page 287 of the *Journal*, issue of August 9, 1934. Eminent physicians from Yale, Harvard, Johns Hopkins, Cornell, New York University and Bellevue Hospital, University of Pennsylvania and Columbia University, will present papers and discussions dealing with interesting medical subjects.

The meeting will be held September 18, 19, and 20, 1934. A copy of the program will be sent on application to Dr. Creighton Barker, 129 Whitney Avenue, New Haven, Connecticut. The registration fee is $2.00.

This is one of the outstanding Congresses of the year, and physicians who do not live in Connecticut may register.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

MINTZ, E. ROSS. M.D. Université de Paris 1928. Assistant in the Genito-Urinary Out-Patient Department, Massachusetts General Hospital. Address: 6 Commonwealth Ave., Boston. Associated with him is

SMITH, GEORGE GILBERT, A.B., M.D. Harvard University School of Medicine 1908. F.A.C.S. Urologist, Massachusetts General Hospital and Palmer Memorial Hospital. Surgeon, Huntington Memorial Hospital. Address: 6 Commonwealth Ave., Boston. Their subject is "Autopsy

Findings in 100 Cases of Prostatic Cancer." Page 479.

LEHNHERR, EARL R. A.B., M.A., M.D. Harvard University School of Medicine 1931. Formerly, House Officer, New England Deaconess Hospital 1930-1931. House Officer, Boston City Hospital, Fifth Medical Service, 1932-1933. Instructor in Biochemistry, University of Kansas, 1925-1928. His subject is "The Value of Icteric Indices and Plasma Lipids in the Diagnosis of Jaundice." Page 487. Address: 472 Commonwealth Ave., Boston.

PENHALLOW, DUNLAP P. S.B., M.D. Harvard University School of Medicine 1906. F.A.C.S. Professor of Clinical Orthopedics, Georgetown University Medical School. Attending Surgeon, Gallinger Municipal Hospital. Surgical Consultant, U. S. Diagnostic Center, Washington, D. C. Surgical Staff, Providence Hospital. Surgical Consultant, Chevy Chase Sanatorium. His subject is "An Unusual Case of Traumatic Hernia." Page 492. Address: 1830 Jefferson Place, N. W., Washington, D. C.

HENDERSON, FRANCIS F. M.D. Tufts College Medical School 1911. A Member of the Harvard Teaching Service at the Boston City Hospital. Visiting Surgeon, Boston City Hospital. Address: 475 Commonwealth Ave., Boston. Associated with him is

FOISIE, PHILIP S. S.B., M.D. Harvard University School of Medicine 1924. Instructor in Surgery, Tufts College Medical School. Assistant in Surgery, Boston City Hospital, Out-Patient Department. Address: 520 Commonwealth Ave., Boston. Their subject is "Report of Case of Torsion of the Spermatic Cord in a Baby Ten Months Old." Page 493.

GRAVES, ROBERT J. B.Sc., M.D. Harvard University School of Medicine 1903. F.A.C.S. Chief Surgeon, Boston and Maine Railroad. Medical Director, United Life and Accident Insurance Company. His subject is "President's Address." Page 495. Address: 5 South State St., Concord, New Hampshire.

DEMING, ROBERT M. M.D. University of Vermont College of Medicine 1916. Superintendent and Medical Director, New Hampshire State Sanatorium, Glencliff, New Hampshire since 1923. His subject is "Pulmonary Tuberculosis. Medical Aspect." Page 500. Address: Glencliff, New Hampshire.

CORRESPONDENCE

CORRESPONDENCE RELATING TO THE EDITORIAL "STARTLING FACTS!"

September 5, 1934.

Managing Editor, *New England Journal of Medicine*,

The editorial "Startling Facts!" in the August 30 issue of the *New England Journal of Medicine* has just come to our attention.

We trust you will permit us to correct the statement that is made in the last two paragraphs of this article.

First—next to the last paragraph—"it claims openly to be accepted by the American Medical Association, Committee on Foods, and by the Wisconsin Alumni Research Foundation."

This is absolutely true.

Secondly—last paragraph—"Recent advice from the Committee on Foods of the American Medical Association, however, states that this particular advertisement was not passed on by the Committee or by the Wisconsin Alumni Research Foundation."

That you may have further proof of the fact that this statement is not true, we are enclosing copy of a letter under date of June 7, 1934 from the Wisconsin Alumni Research Foundation which speaks for itself.

Under the circumstances and in view of the fact, as you personally told me yourself, that any product advertised in the *Journal* practically carries the endorsement of the same and in view that the *Journal* is carrying this advertising of Whiting's Irradiated Vitamin 'D' Milk monthly, it would seem only fair to all concerned that you should correct this statement in the first forthcoming issue possible.

Sincerely,

A. E. GREENLEAF,
President, The Greenleaf Co.
Advertising and Merchandising Counsel,
Union Savings Bank Building,
216 Tremont Street,
Boston, Mass.

EDITORIAL CORRECTION

In reference to the editorial "Startling Facts!" published on August 30, the *Journal* was in error in stating that the Whiting Milk Companies advertisement in the *Boston Evening Transcript* of July 21 was not endorsed by the Wisconsin Alumni Research Foundation. It apparently was endorsed by the Foundation, but when its nature was called to their attention the advertisement was withdrawn. In confirmation of the reference to the action of the American Medical Association, the letter of Dr. Morris Fishbein is appended.

This whole discussion raises the question of how much responsibility an association or a foundation assumes in endorsing advertising material. It would seem to us as if such endorsement carried with it a distinct obligation to know accurately and in detail the nature of all such material employed; and also an obligation to insist that its propriety be beyond question.

August 8, 1934.

Dr. Morris Fishbein,
The Journal of the American Medical Association,
535 North Dearborn Street,
Chicago, Illinois.

Dear Dr. Fishbein:

I appreciate your note of July 31st, stating that the advertisement that we questioned has been with-

drawn by the Wisconsin Foundation, but that scarcely answers my question.

If you will recall that advertisement, it stated that the American Medical Association accepted the advertisement. What I should like very much from you, as soon as possible, is a definite answer: did the American Medical Association accept this advertisement? If you are unwilling to answer that question, I must assume that the American Medical Association did accept that advertisement and have laid themselves open to very serious criticism.

I hope to have an immediate answer from you. It is a serious matter to have a firm of Whiting's stand make such a statement if it is not so. If it is so, the blame of course comes upon the American Medical Association, and therefore we have a right to criticize and object to any such careless, untruthful advertisements being accepted by the Association.

I should appreciate an early reply from you.

Sincerely yours,

ROBERT L. DENORMANDIE.

THE JOURNAL OF THE
AMERICAN MEDICAL ASSOCIATION
535 North Dearborn Street
Chicago

August 17, 1934.

Dr. Robert L. DeNormandie,
Boston, Massachusetts.

Dear Doctor DeNormandie:

A memorandum from Mr. R. Hertwig, Secretary of the Committee on Foods, reads as follows:

"The particular Whiting newspaper advertisement was not passed on by the Committee or this office.

"The Committee Rules require only that copy of all pieces of advertising be submitted to its office. There is no requirement that the advertising be passed on before it is published, which would be a practical impossibility. Objectionable advertising is passed on after issuance and the sponsors are advised of criticisms. If subsequent advertising does not indicate compliance with these criticisms, then action is taken to withdraw acceptance.

"Although by this procedure some inappropriate advertising is permitted to come before the public, it is quickly detected and stopped."

The Wisconsin Alumni Research Foundation immediately withdrew the advertising when the matter was called to their attention. In passing on products the Committee on Foods indicates what are considered to be acceptable claims from a medical point of view. The Wisconsin Alumni Research Foundation has now been informed that quotations even from alleged authorities must be submitted before they will be considered acceptable as advertising.

Very truly yours,

MORRIS FISHBEIN.

RECENT DEATHS

OSGOOD—GILMAN OSGOOD, M.D., of Rockland, Massachusetts, died at his home, September 8, 1934, after an illness of several weeks. He was born in Abington in 1863, the son of Gilman and Isabella (Foster) Osgood. As a lad he worked in his father's factory. His early education was acquired in the public schools of Abington, and he graduated from the Bellevue Hospital Medical School in 1886. After graduating, he served in the King's County Hospital, Brooklyn, and after a year was appointed on the staff of the King's County Hospital for the Insane; after two years he became assistant superintendent of the King's County Hospital.

In 1890 he engaged in practice in Rockland, and was appointed medical examiner in 1906. He held this position at the time of his death. Dr. Osgood joined the Massachusetts Medical Society in 1892, and was also a Fellow of the American Medical Association. He was president of the Massachusetts Medico-Legal Society, and was the founder of the Hatherly Medical Society. He was also a member of the Massachusetts Association of Boards of Health. He was affiliated with the Masons, Odd Fellows, and other organizations.

Dr. Osgood is survived by his widow, Mrs. Mabel (Russell) Osgood, and four children, Mrs. Louise Wales of Abington, Mrs. Ellen Evans of New York, Russell Osgood of Rockland, and Foster Osgood of Lexington.

PEARSON—CHARLES LUSBY PEARSON, M.D., of 106 Washington Street, Newton, Massachusetts, died at his home September 8, 1934. Dr. Pearson was born in 1860, the son of Samuel and Sarah (Pell) Pearson, at Lincoln, Ill. He graduated from the University of Maryland Medical School in 1883, joined the Massachusetts Medical Society in 1902 and retired in 1932.

He is survived by his widow, two sons, a brother, and two sisters.

LOUGHLIN—JOHN JOSEPH LOUGHLIN, M.D., aged 54, of Richardson Avenue, Wakefield, Massachusetts, died September 6, 1934, following a "shock." He received his M.D. degree from the Boston University Medical School in 1903, joined the Massachusetts Medical Society in 1923, and had practiced in Wakefield for the past twenty-seven years.

He is survived by his widow; three sons, Leo, John, and Paul; two brothers, Thomas P., and Jeremiah F. Loughlin; and a sister, Mrs. W. Arthur Robbins, all of Wakefield.

TRUEMAN — HARMON SILAS TRUEMAN, M.D., of West Medford, Massachusetts, died recently in the Trull Hospital, Biddeford, Maine. He left his prac-

tice August 1, for a visit at his summer home at Cape Porpoise, Maine.

Dr. Trueman was born in 1858, and graduated in medicine from the University of Pennsylvania Medical School in 1880. He formerly practiced in Somerville and moved to West Medford in 1926.

He joined the Massachusetts Medical Society in 1893, and resigned in 1924. He was also a Fellow of the American Medical Association.

Dr. Trueman is survived by his widow, three daughters, Mrs. S. E. Armstrong of Dumont, N. J.; Miss Muriel Trueman and Miss Hazel Trueman of West Medford, and a son, Alfred S. Trueman of New York City.

SHEEHAN—MARTIN DAVID SHEEHAN, M.D., of 34 Pleasant Street, Stoneham, died at the Winchester Hospital, September 4, 1934, following an automobile accident. The report of his death contains the assumption that he was the victim of a "heart attack" which explained the cause of the accident.

He was born in Brookline, Massachusetts, in 1871, the son of David H., and Mary Sheehan. He was educated in the public schools of Brookline, and took courses at the Harvard Medical School, and about five years ago went to Vienna for further study.

He joined the Massachusetts Medical Society in 1897. He was formerly a member of the Board of Health of Stoneham.

Dr. Sheehan is survived by his widow, Mrs. Jeanette Sheehan; two sons, George D., and Robert; a daughter, Mrs. Kathleen Morrill of California; a sister, Dr. Helen T. Sheehan; and a brother, Edward T. Sheehan.

NOTICES

CARNEY HOSPITAL GOLF TOURNAMENT

The Fourth Annual Carney Hospital Golf Tournament will be held at the Commonwealth Country Club, September 19, 1934.

A team match between the Carney and Boston City Hospital Staffs, will be held for the second leg of the Silver Trophy. The Tournament will be followed by dinner at the Club House, with distribution of prizes and entertainment. All doctors are invited.

ROY J. HEFFERNAN,
Chairman of Golf Committee.

REMOVALS

DONALD S. KING, M.D., announces the removal of his office to the Massachusetts General Hospital, Boston.

WALTER S. BURRAGE, M.D., wishes to announce the removal of his office to 330 Dartmouth Street. Telephone Kenmore 2640.

NOTICES OF MEETINGS

THIRTEENTH ANNUAL CONGRESS OF ANESTHETISTS

BOSTON, MASS., OCTOBER 15-19, 1934
(Clinical Congress of Surgeons Week)

PRELIMINARY PROGRAM

Associated Anesthetists of the United States and Canada
International Anesthesia Research Society
Eastern Society of Anesthetists
Mid-Western Association of Anesthetists

Headquarters, Sessions and Exhibits
Grand Ball Room and Lounges
The Hotel Bradford of Boston
Tremont, Opposite Hollis Street

Fellows of the American College of Surgeons and members of the Boston and New England Surgical Societies, Norfolk and Suffolk District Medical Societies, Massachusetts Dental Society (Metropolitan District) and New England Dental Society are most cordially invited to attend any and all of the sessions of the Congress of Anesthetists.

OFFICERS OF THE CONGRESS

Associated Anesthetists of the United States and Canada

Charles J. Wells, M.D., Syracuse, N. Y., President-Elect.

J. Robert Burrows, M.D., San Francisco, 1st Vice-President.

Edmund Lunney, M.D., St. John, N. B., Canada, 2nd Vice-President.

John Hayward, M.D., Providence, R. I., 3rd Vice-President.

Henry W. Kearney, M.D., Washington, D. C., 4th Vice-President.

F. H. McMechan, M.D., Rocky River, Ohio, Secretary-General.

International Anesthesia Research Society

Honorary Officers and Advisory Board

Honorary President: Professor Wm. deB. MacNider, M.D., Sc.D., University of North Carolina, Chapel Hill, N. C.

Eastern Society of Anesthetists

John A. Hayward, M.D., Providence, R. I., President.

Henry S. Ruth, M.D., Philadelphia, Pa., Vice-President.

Chas. J. Wells, M.D., 932 S. Salina St., Syracuse, N.Y., Secretary-Treasurer.

Mid-Western Association of Anesthetists

J. Stanley Gardner, M.D., Lakewood, Ohio, President-Elect.

B. H. Harms, D.D.S., 826 Medical Arts Bldg., Omaha, Neb., Secretary-Treasurer.

Boston Society of Anesthetists

Philip D. Woodbridge, M.D., Boston, President.
Russell F. Sheldon, M.D., 31 Pinckney Street, Boston, Secretary-Treasurer.

PROGRAM

MONDAY MORNING, OCTOBER 15
Special Demonstration 9 A.M.
Massachusetts General Hospital.

Through the courtesy of H. H. Bradshaw, M.D., in charge of anesthesia and Monica Harnden, M.D., Resident Anesthetist, there will be a Special Demonstration involving some of the anesthetic problems in open chest surgery with the use of basal avertin and intratracheal gas-oxygen-ether anesthesia. This demonstration will be staged in one of the laboratories of the Surgical Department of the Massachusetts General Hospital.

Anesthesia Clinics 9 A.M. - 12 M.
At Various Boston Hospitals

Through the coöperation of the Officers and Members of the Boston Society of Anesthetists and a special committee with Philip D. Woodbridge, M.D., as chairman, Anesthesia Clinics will be held in the following hospitals—Beth Israel Hospital, Boston City Hospital, Faulkner Hospital, Massachusetts General Hospital, Massachusetts Memorial Hospitals, New England Deaconess Hospital and New England Baptist Hospital. Those in attendance at the Congress are requested to plan in advance for attending the Special Demonstration and Anesthesia Clinics. Further details are given in the Directory of Anesthesia Clinics and Laboratory Demonstrations in this program.

MONDAY AFTERNOON, OCTOBER 15
Grand Ball Room and Lounges, Bradford Hotel
Opening Session 2 P.M.

F. H. McMechan, M.D., Secretary-General, Presiding

Address of Welcome—Harold G. Giddings, A.B., M.D., Visiting Surgeon Whidden Memorial Hospital, Everett, Mass.

Progress in the Specialty

J. Stanley Gardner, M.D., Mid-Western President, Presiding

Horace Wells, (Presidential Address, Associated Anesthetists of the United States and Canada)—Charles J. Wells, M.D., Secretary, Eastern Society of Anesthetists; Department of Anesthesia, University of Syracuse Medical School, Syracuse, N. Y.

Hospital Anesthesia Service—(Presidential Address, Eastern Society of Anesthetists)—John A. Hayward, M.D.

The Boston Anesthesia Service—William Temple, Jr., M.D., Anesthetist, Boston, Mass.

Anesthesia from the Layman's Viewpoint, (Presidential Address, Mid-Western Association of Anesthetists)—J. Stanley Gardner, M.D.

Some Medico-Legal Aspects of the Nursing Anesthesia Problem—W. Chalmers-Francis, M.D.

MONDAY EVENING, OCTOBER 15

Grand Ball Room and Lounges, Bradford Hotel
International Fellowship Session 8 P.M.
Charles J. Wells, M.D., Associated President, Presiding

Intrathecal Nerve Root Block: Some Contributions and a New Technique, (British Address: Motion Picture Demonstration)—W. Etherington-Wilson, F.R.C.S.

Modern Methods, Accessible to the Anesthetist, for the Treatment of Pain, (Italian Address: Motion Picture Demonstration)—A. Mario Dogliotti, M.D.

Preoperative Determination of Individual Susceptibility in Relation to the Choice of the Method of Anesthesia, (Italian Address)—Professor A. Trivellini.

TUESDAY MORNING, OCTOBER 16

Anesthesia Clinics 9 A.M. - 12 M.
At Various Boston Hospitals

Through the coöperation of the Officers and Members of the Boston Society of Anesthetists and a special committee with Philip D. Woodbridge, M.D., as Chairman, Anesthesia Clinics will be held in the following hospitals—Beth Israel Hospital, Boston City Hospital, Faulkner Hospital, Massachusetts General Hospital, Massachusetts Memorial Hospitals, New England Deaconess Hospital and New England Baptist Hospital. It is expected that operation and anesthesia schedules for the various hospitals can be posted on the Congress bulletin board on Monday night.

TUESDAY AFTERNOON, OCTOBER 16

Grand Ball Room and Lounges, Bradford Hotel

Business Session

Associated Anesthetists, Research Society and Midwestern Anesthetists,
1:30 to 2 P.M.

Current Researches in Anesthesia and Analgesia,
2 P.M.

Prof. Wm. deB. MacNider, Hon. I. A. R. S. President, Presiding

A Consideration of the Susceptibility and the Resistance of Tissue Cells to the General Anesthetics, (Hon. Presidential Address), International Anesthesia Research Society. (Lantern Slide Demonstration)—William deB. MacNider, M.D., Sc.D., LL.D.

I. Reflex Closure of the Glottis by Stimulation of Afferent Visceral Nerves. II. Hemodynamic and Respiratory Changes Following Manipulation and Traction of the Gastrohepatic Ligament—Professor Arno B. Luckhardt, M.D., Nathan Brewer, B.S., D. S. Bryant, B.S., and W. M. Lees.

The Stability of U. S. P. Ether After the Metal Container Has Been Opened—Harry Gold, M.D., and David Gold, B.A.

Further Studies in the Effects of Anesthetics on the Kidneys—Professor M. S. Dooley, M.D.

The Influence of Temperature and the Effect of Certain Sedatives on the Oxygen Requirement—Professor Allan Winter Rowe, Ph.D.

TUESDAY EVENING, OCTOBER 16

Grand Ball Room and Lounges, Bradford Hotel
International Fellowship Session, 8 P.M.

John A. Hayward, M.D., Eastern President, Presiding

Anesthetic Methods and Difficulties in South Africa, (South African Address)—Eric G. Van Hoogstraten, B.A., M.B., Ch.B.

Anesthesia in Obstetrics with Special Reference to Complicated Cases, (Canadian Address)—Samuel Johnston, M.A., M.D., C.M., F.A.C.P.

The Clinical Value and Practical Results of Evipan-Sodium Intravenous Anesthesia, (Hungarian Address)—Deszo Hudovernig, M.D., Budapest, Hungary.

Intravenous Evipan Anesthesia, (British Address)—Ronald Jarman, D.S.C., M.R.C.S., (Eng); and A. Lawrence Abel, M.S., (Lond.), F.R.C.S.

WEDNESDAY MORNING, OCTOBER 17

Laboratory Demonstration 9 A.M.
Evans Memorial Institute

Through the courtesy of Professor Allan Winter Rowe, Ph.D., special laboratory demonstrations and talks will be given by Professor Rowe and his associates of the Evans Memorial Institute and among the subjects to be discussed and demonstrated may be mentioned (1) Oxygen Metabolism; (2) Blood Gases; (3) Mechanical Heart; and (4) Cardiac Oscillograph.

WEDNESDAY AFTERNOON, OCTOBER 17

Dental and Oral Surgery Anesthesia Clinics
2 to 5 P.M. Forsyth Infirmary

Through the courtesy of the Massachusetts Dental Society (Metropolitan District) and its special committee, William M. Parks, D.D.S., Chairman, Frank W. Rounds, D.D.S., and Edwin J. Morse, D.D.S., operative and dental anesthesia clinics will be held at the Forsyth Infirmary. In the large operative clinic room, members of the Forsyth Staff and other Boston dentists will demonstrate such operations as the preparing of sensitive cavities, root amputations, surgical treatment of pyorrhea, etc., under local anesthesia. In the surgical amphitheatre ethyl chlorid anesthesia will be demonstrated by the Forsyth Staff for the removal of deciduous teeth of children; and gas-oxygen anesthesia, in its various agents and combinations, will be demonstrated for routine extractions, impacted teeth and also for minor oral surgery. Busses, provided by the Metropolitan District Dental Society will take those desiring to attend, to the Forsyth Infirmary, leaving the Hotel Bradford at 1:45 P.M. sharp and returning at about 5:30 P.M.

WEDNESDAY EVENING, OCTOBER 17

Buffet Supper, 6-8 P.M.
Lobby Salon, Hotel Bradford

A joint buffet supper will be held in the Lobby Salon of the Hotel Bradford and members of the Metropolitan District Dental Society, the New England Dental Society and the Congress of Anesthetists are invited to attend.

WEDNESDAY EVENING, OCTOBER 17

Grand Ball Room and Lounges, Bradford Hotel
Joint Guest Session with the Massachusetts Dental Society (Metropolitan District)

Anesthesia in Dentistry and Oral Surgery 8 P.M.

William M. Parks, D.D.S., Hon. Chairman, Presiding

Evaluation of Risks for Dental and Oral Surgery—John B. Woodman, D.D.S.

Conscientious, Intelligent Anesthetization with Nitrous Oxid-Oxygen for Dental Surgery, (Motion Picture Demonstration)—W. Harry Archer, D.D.S.

THURSDAY MORNING, OCTOBER 18

Special Laboratory Demonstration 9 A.M.
Massachusetts General Hospital and
Harvard Medical School

Through the courtesy of H. H. Bradshaw, M.D., in charge of Anesthesia Service and Monica Harnden, M.D., Resident Anesthetist, a special laboratory demonstration in the Surgical Department of the Massachusetts General Hospital will be given showing the effects of tracheal obstruction and anoxemia on pulmonary circulation and respiratory obstruction. There will also be other demonstrations on respiratory physiology at the Harvard Medical School.

THURSDAY AFTERNOON, OCTOBER 18

Grand Ball Room and Lounges, Bradford Hotel

Business Session

Eastern Society of Anesthetists
1:30 to 2 P.M.

Complicating Factors of Anesthesia and Operation, 2 P.M.

J. Stanley Gardner, M.D., Mid-Western President, Presiding

Estimation of and Methods of Meeting Surgical and Anesthetic Risks and Postoperative Complications in Surgical Diseases of the Biliary Tract—Frederic Vinal Hussey, M.D.

Studies in Water Requirements of Surgical Patients—Frederick A. Coller, B.S., M.S., M.D., and Walter G. Maddock, A.B., M.S., M.D.

Anesthetic Deaths

The Relationship of Anesthesia to Operative Fatalities: A Study of Surgical Deaths at the Massachusetts General Hospital—Russell F. Sheldon, M.D.

Further Studies in Sensitization to General and Local Anesthesia—George L. Waldbott, M.D.

Basal Narcosis

Results in the Use of Avertin in Over 4,000 Cases at the Boston Children's Hospital—Patrick J. Mahoney, M.D.

THURSDAY EVENING, OCTOBER 18

Lobby Salon, Bradford Hotel
Annual Dinner 7 P.M.
$2.50 a Cover

Be sure and bring your Ladies to enjoy this auspicious occasion and also invite your Surgeon and Specialist Friends and their Ladies to attend. Special Honors will be bestowed for Meritorious Researches, Contributions to the Literature and Services to the Specialty.

FRIDAY MORNING, OCTOBER 19

Grand Ball Room and Lounges, Bradford Hotel
Therapeutic Use of Anesthetics and Gases, 9 A.M.
John A. Hayward, M.D., Eastern President, Presiding

The Value of Ether and Chloroform Narcosis in the Treatment of Cancer—Robert Benner, M.D.

Indications for Oxygen Therapy in Respiratory Diseases—John H. Evans, M.D., and Clarence J. Durshordwe, M.D.

The Treatment of Heart Failure by Continuous Oxygen Therapy—Alvan L. Barach, M.D.

Denarcotization

Experiments in Denarcotization and Resuscitation —Paul M. Wood, B.S., M.D.

Further Research Studies in Palinesthesia—Walter V. MacGilvra, D.D.S.

FRIDAY AFTERNOON, OCTOBER 19

Grand Ball Room and Lounges, Bradford Hotel
Newer Anesthetics and Methods 2 P.M.
Chas. J. Wells, M.D., Associated President, Presiding

Spinal Anesthesia for Chest Surgery — Harry J. Shields, B.A., M.B.

The Effect of Oxygen in the Prevention of the Liver Necrosis Produced by Volatile Anesthetics—I. S. Ravdin, M.D., Samuel Goldschmidt, Ph.D., and Baldwin Lucke, M.D.

Some Aspects of Vinyl Oxid (Vinesthene) Anesthesia —Wesley Bourne, M.D., M.Sc., F.R.C.P.

Cyclopropane: Further Researches, Experiences and Results—Prof. Erwin R. Schmidt, M.D., F.A.C.S., and Ralph M. Waters, M.D.

A Study of the Merits and Disadvantages of the Absorption of Carbon Dioxid in General Anesthesia—E. I. McKesson, M.D., F. W. Clement, M.B., K. C. McCarthy, M.D.

OTHER IMPORTANT MEETINGS

Southern Association of Anesthetists,
San Antonio, Texas, November 14-15, 1934.
Hotel St. Anthony

The Southern Anesthetists will revive their meetings with the Southern Medical Association in San Antonio, Texas, November 14-15, 1934. Those interested in presenting papers or in attending are requested to get in touch with William Hamilton Long, M.D.. Secretary, 433 Francis Bldg., Louisville, Ky.

FURTHER DETAILS

Transportation—As usual the one and a half round trip fare will be available for the Congress under the auspices of the College of Surgeons and the Associated Anesthetists on the Certificate plan. Get your Certificate from your Ticket Agent at the time of buying your full fare transportation to Boston. On validation by the proper railway authority at the Exhibits of the Congress of Surgeons at the Copley-Plaza and Hotel Statler, your Certificate will entitle you to half-fare transportation home over the same route you came by. If you prefer bus or airplane travel, inquire for rates and make your reservations well in advance.

Ladies Entertainment—Visiting and local ladies are requested to register with the Secretary-General in the Grand Ball Room, so that they may have full information about the entertainment that is being planned for them. Boston with its wealth of historical interest will offer every possible opportunity for a wonderful time.

DIRECTORY OF CLINICS

Beth Israel Hospital, 330 Brookline Ave., Boston. Same directions as for the Deaconess Hospital. Boris Rapoport, M.D., Anesthetist, and assistants. Operations every day from 8 A.M. to 12 M. Methods used, gas-oxygen (circle filter), ether, regional and spinal. All surgical departments will participate.

Boston City Hospital, 818 Harrison Ave., Boston. t is 1.3 miles from the Hotel Bradford. In taxi or own car proceed south up Shawmut Ave., one mile and urn east at Concord St., proceed one-fourth mile. This hospital may also be reached by entering Lagrange St. subway station and taking elevated train south, getting out at North Hampton St. Station and turning east one block to Harrison Ave. Frank W. Marvin, M.D., Wm. A. Noonan, M.D., and S. C. Wiggin, M.D., Anesthetists. Six surgical services, including orthopedic service, operating daily schedule. Methods used, spinal anesthesia with pantocain, procain, aminocain, metycain and nupercain; intravenous evipan sodium and nembutal; inhalation vinesthene; and rectal avertin.

Faulkner Hospital, 1154 Center St., Jamaica Plain. By taxi or own car, Tremont St. to Stuart, left on Stuart to Huntington Ave., left on Huntington to Riverway, Route 1, left on Riverway and continue on

Route 1 to hospital. Or by street car at Boylston St. subway station, Tremont and Boylston, taking Jamaica, Arborway car via S. Huntington Ave. to Soldiers Monument in Jamaica Plain at Eliot St., getting transfer to W. Roxbury bus which passes entrance to hospital. Sidney C. Wiggin, M.D., John H. Holleran, M.D., Charles Sheldon, M.D., and Alexander Aitken, M.D., Anesthetists. Operating schedule from 8 A.M. to 12 M. every day. Methods used, nitrous oxid-oxygen-ether, basal avertin, and spinal with novocain and pantocain. Semi-closed technique with towel cone, nitrous oxid with carbon dioxid induction, carbon dioxid absorption method of gas-oxygen without ether. Also premedication drugs.

Massachusetts General Hospital, Grove and Fruit Sts., Boston. In taxi or own car drive north on Tremont St. to Boylston, turn left on Boylston, turn right at first stop light which is Charles St., continue on Charles St. to the traffic circle and at this point bear right on Cambridge St. and take first left turn on North Grove St. North Grove St. will bring you directly into the yard of the Massachusetts General Hospital. Or walk north on Tremont St. one block, turn left on Stuart St. one block and continue on Eliot St., one block to Park Square. Take bus marked Bowdoin Square at monument in Park Square, get off at North Grove St., and walk north one block. H. H. Bradshaw, M.D., in charge of Department of Anesthesia and Monica Harnden, M.D., Resident Anesthetist. Operative schedules from 9 A.M. to 5 P.M. Special demonstration Monday 9 A.M. on anesthetic problems in open chest surgery with use of basal avertin and intratracheal gas-oxygen-ether. Special demonstration Thursday 9 A.M. showing the effects of tracheal obstruction and anoxemia on pulmonary circulation and respiration.

Massachusetts Memorial Hospitals, 750 Harrison Ave., Corner of Stoughton St., next building but one to Boston City Hospital. Same directions as for Boston City Hospital. Mary A. Leavitt, M.D. and Eleanor B. Ferguson, M.D., Anesthetists. Operating schedule, general surgery, 9 A.M. to 12:30 P.M. Orthopedic, nose, throat and ear, Monday, Wednesday and Friday, genito-urinary Tuesday, Thursday and Saturday. Methods used, open drop ether gas-oxygen with and without carbon dioxid absorption, basal avertin, spinal and epidural with novocain.

New England Deaconess Hospital, 16 Deaconess Road, Boston. By taxi or own auto. North on Tremont St., turn left on Stuart and immediately fork and bear left on Stuart St. Turn right on Dartmouth St. after four blocks, turn left on Commonwealth Ave. to Kenmore Square junction of Commonwealth and Brookline Aves., and Beacon St. Turn left on Brookline Ave. to Deaconess Road, turning right. Or walk north on Tremont St. three blocks to Boylston St., take subway car marked "Lake St., Commonwealth" or "Watertown" or "Reservoir-Beacon" to Kenmore Square. Transfer to bus marked "Chestnut Hill" out Brookline Ave., to Deaconess Road. Lincoln F. Sise, M.D., Philip D. Woodbridge, M.D., and Urban H. Eversole, M.D., Anesthetists. Operating schedule, general surgery including thyroid surgery; orthopedic, nose, throat and genito-urinary, 8 A.M. to 1 P.M. Brain surgery, 1 P.M. when scheduled. (All Lahey Clinic Cases.) Methods used, general anesthesia with nitrous oxid, ethylene, cyclopropane and ether by the carbon dioxid absorption or intratracheal techniques and pharyngeal insufflation; basal avertin; intravenous sodium-evipan, spinal novocain and rectal metycain.

New England Baptist Hospital, 85 Parker Hill Ave., (Roxbury), Boston. By taxi or own car, north on Tremont St., turn left on Stuart St., immediately fork bearing left on Stuart St. to end of street, bear left on Huntington Ave., turn left on Parker Hill Ave. to hospital on farther corner of Fisher Ave. Or walk north on Tremont St. three blocks to corner of Boylston St. Take subway car marked "Cypress St." or "Arborway" out Huntington Ave. Get off at Parker Hill Ave., walk up Parker Hill Ave., to Baptist Hospital on farther corner of Fisher Ave. Anesthetists, operating schedule and methods used same as for New England Deaconess Hospital.

IMPORTANT NOTICE

Remember the banquet and meetings of the INTERNATIONAL ASSOCIATION OF INDUSTRIAL ACCIDENT BOARDS AND COMMISSIONS to be held at the Hotel Statler, Boston. Meetings are scheduled from September 24 to September 28.

NEW ENGLAND SURGICAL SOCIETY

The Annual Meeting of the New England Surgical Society will be held in the Flemming Museum, Colchester Avenue, Burlington, Vermont, on Friday, September 28 and Saturday, September 29.

GENERAL PROGRAM

FRIDAY, SEPTEMBER 28, 1934

8 A.M. Registration and Information. Flemming Museum.
9 A.M. to 1 P.M. Scientific Program. Flemming Museum.
1:30 P.M. Luncheon at Burlington Country Club, South Prospect Street.
3 P.M. Sail on Lake Champlain. Boat leaves foot of King Street.
7 P.M. Assemble at Roof Garden. Hotel Vermont.
7:30 P.M. Annual Dinner. Hotel Vermont.

SATURDAY, SEPTEMBER 29, 1934

9 A.M. Annual Meeting of the Society. Flemming Museum.
9:30 A.M. to 1:30 P.M. Scientific Program.

SCIENTIFIC PROGRAM

FRIDAY MORNING, SEPTEMBER 28

1. "A Two-Stage Operation for Bladder Tumors." Dr. Arthur H. Crosbie.
   Discussion: Dr. William G. Townsend, Dr. Clyde L. Deming.

2. "The Value of Prolonged Preoperative Drainage in Prostatectomy." Dr. J. Dellinger Barney.
   Discussion: Dr. Winford O. Wilder, Dr. Alfred Mitchell, Jr.
3. "Tidal Drainage in the Treatment of the Atonic Urinary Bladder with Transverse Myelitis." Dr. Donald Munro; Dr. Joseph Hahn—by invitation.
   Discussion: Dr. William J. Mixter, Dr. Harry C. Clifton.
4. "Indication for and Results of Total Cystectomy for Cancer of the Bladder." Dr. William C. Quinby.
   Discussion: Dr. Richard F. O'Neil, Dr. George G. Smith.
5. "Uretero-rectal Anastomosis." Dr. Thomas N. Hepburn.
   Discussion: Dr. Frank H. Lahey, Dr. Ernest G. Crabtree.
6. "The Association of Pylephlebitis and Appendicitis." Dr. Arthur W. Allen; Dr. W. H. Snyder, Jr., Dr. M. G. Hall—by invitation.
   Discussion: Dr. Archibald M. Fraser, Dr. Robert C. Cochrane.
7. "Recurrent Peripheral Thrombophlebitis Causing Pulmonary Embolism." Dr. John Homans.
   Discussion: Dr. Donald S. Adams, Dr. Robert J. Graves.
8. "Acute Haematogenous Osteomyelitis." Dr. Richard H. Miller.
   Discussion: Dr. Robert B. Osgood, Dr. H. Bertram Lambert.

SATURDAY MORNING, SEPTEMBER 29

1. "The Limitations of Enterostomy and Undesirable Effects Incident to Its Use." Dr. Isaac M. Webber.
   Discussion: Dr. Edward H. Risley, Dr. Albert A. Barrows.
2. "Slipping Epiphysis of the Head of the Femur." Dr. James W. Sever.
   Discussion: Dr. Allan Woodstock, Dr. Ezra A. Jones.
3. "Fractures of the Femoral Neck Treated by Blind Nailing." Dr. Ernest L. Hunt; Dr. John W. O'Meara—by invitation.
   Discussion: Dr. Frederic J. Cotton, Dr. Carleton R. Metcalf.
4. "Traumatic Rupture of the Diaphragm Resulting From a Shell Fragment." Dr. Walter Phippen, Dr. Philemon E. Truesdale.
   Discussion: Dr. Wyman Whittemore, Dr. Horace Binney.
5. "Technique of the Resection of the Cecum by a One-Stage Procedure." Dr. Samuel C. Harvey.
   Discussion: Dr. Ernest M. Daland, Dr. Daniel F. Jones.
6. "Vaginal Hysterectomy." Dr. Oliver N. Eastman.
   Discussion: Dr. Charles L. Larkin, Dr. Frank A. Pemberton.
7. "Gynecologic Problems of Interest to the Surgeon in General Practice." Dr. Arthur H. Morse.
   Discussion: Dr. Stephen Rushmore, Dr. Henry T. Hutchins.
8. "Bleeding Gastric Ulcer." Dr. George A. Moore.
   Discussion: Dr. David Cheever, Dr. Walter C. Seelye.

INTERNATIONAL ASSEMBLY OF THE INTER-STATE POSTGRADUATE MEDICAL ASSOCIATION OF NORTH AMERICA

The International Assembly of the Inter-State Postgraduate Medical Association of North America will be held in the Public Auditorium, Philadelphia, Pennsylvania, November 5-9, 1934. Many distinguished teachers and clinicians will appear on the program. A major list of the names of the contributors to the program, with other information, appears on page vii of the Advertising Section of this *Journal*. All members of the Massachusetts Medical Society are cordially invited to attend. Registration fee of $5.00 admits all members of the profession in good standing.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

September 18, 19 and 20—Tenth Clinical Congress of the Connecticut State Medical Society. See page 287, issue of August 9.

September 19—The Carney Hospital Golf Tournament. See page 515.

September 24-28—Convention of the International Association of Industrial Accident Boards and Commissions. See page 473, issue of September 6.

September 28-29—New England Surgical Society will meet at Burlington, Vermont. See page 519.

October—International Medical Postgraduate Courses. See page 335, issue of August 16.

October 7-20—Sixth Annual Medico-Military Symposium, at the Mayo Clinic. See page 382, issue of August 23.

October 8, 9 and 10—Association of Military Surgeons of the United States. See page 473, issue of September 6.

October 15-19—Sixth Annual Session of the Association of Record Librarians of North America at Hotel Kenmore, Boston. Chairman of Committee on Arrangements—Miss Edith M. Robbins, Peter Bent Brigham Hospital, Boston.

October 15-19—American College of Surgeons at Boston. See page 424, issue of August 30.

October 15-19—Thirteenth Annual Congress of Anesthetists. See page 515.

October 17 — William Harvey Society. Speaker: Dr. Emil Novak of Baltimore.

October 22 - November 2—1934 Graduate Fortnight of the New York Academy of Medicine. See page 1240, issue of June 7.

October 31 - November 2—Massachusetts State Nurses' Association, Hotel Statler, Boston. For information write Miss Helene G. Lee, R.N., 420 Boylston Street, Boston.

November 5-9—International Assembly of the Inter-State Postgraduate Medical Association of North America. See notice above.

April 29 - May 3, 1935—The American College of Physicians will meet in Philadelphia. For information address Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

June, 1935—Medical Library Association will meet in Rochester, N. Y. For details address the Secretary: Miss Frances N. A. Whitman, Librarian, Harvard University Schools of Medicine and Public Health, Boston, Mass.